The Boston Medical and Surgical Journal

TABLE OF CONTENTS

March 16, 1922

THE NEW ENGLAND SURGICAL SOCIETY

Acute Pancreatitis. *By Daniel Fiske Jones, M.D., F.A.C.S.,* Boston 337

ORIGINAL ARTICLES

The Indications for Operation in the Treatment of Injuries Involving the Brain. *By Donald Munro, M.D.,* Boston 242
Concerning the Type of Injury to Renal Epithelial Cells Which Increases the Susceptibility of the Cells to the Action of the General Anesthetics. *By Wm. de B. MacNider, M.D.,* Chapel Hill, N. C. 350
Acute Puerperal Inversion, with the Report of a Case. *By Joseph P. Cohen, M.D.,* Boston 352

CURRENT LITERATURE DEPARTMENT

Bastedo's Symptom in Appendicitis 357
So-Called Abacterial Renal Pyuria 357
Carcinoma of the Prostate 358
Histology and Mortality in Cases of Tumor of the Bladder 358
Synovial Membrane Tumors of Joints 358
Sacral Anaesthesia in Urology 358

EDITORIALS

The Present Status of Pathology 359
A Physician in the Cabinet 361
Mid-Winter Clinics of the Maine Public Health Association and the Maine Medical Association 361
News Items 361

THE MASSACHUSETTS MEDICAL SOCIETY

Membership Changes from January 1, 1922, to March 1, 1922 362

MISCELLANY

Legislative Matters 365
Meeting of the Association of Assistant Physicians of Massachusetts Department of Mental Diseases 367
Department of Public Health Laboratory in the Western Part of the State 367
Deaths from Lethargic Encephalitis ("Sleeping Sickness") 367
National Legislation 368
The Tuberculosis Fight 368
A High Death Rate in Scotland 368

OBITUARY

Joseph Ward Battershall, M.D. 369

CORRESPONDENCE

An Osteopathic Diagnosis 369

The New England Surgical Society

ACUTE PANCREATITIS.*

By Daniel Fiske Jones, M.D., F.A.C.S., Boston.

"Acute Pancreatitis," the title of this paper, is a term which will probably always be used, but I shall try to show, farther along in the paper, that it is not specific enough and that the two terms—acute pancreatic necrosis, or acute hemorrhagic necrosis of the pancreas, and acute interstitial pancreatitis—will describe more accurately the exact condition we are talking about.

So much work has been done on experimental pancreatitis, one feels that it should be quite well understood by this time; but, unfortunately, we seem almost as far from a solution of the problem as ever.

In 1856, Claude Bernard first produced pancreatic necrosis, experimentally, by the injection of bile and sweet oil into the pancreatic duct. Since then, Opie[1], Flexner[2], Hlava[3], Guleke[4], and Brocq[5], and many others, have produced pancreatic necrosis by the injection of various substances into the duct, or gland, itself. The more important of these substances are: gastric and duodenal contents, weak acids and alkalies, fatty acids, normal bile, bile salts, and infected bile.

In 1901, Opie[1] injected bile into the pancreatic duct and produced necrosis. The same year, he published the autopsy report of a case of pancreatic necrosis in which a small stone was found blocking the papilla of Vater in such a way that it converted the biliary and pancreatic systems into one. This supposedly caused a backing up of bile into the pancreatic duct and so caused the pancreatic necrosis. There have been several such cases reported since.

Brocq[6], in 1914, demonstrated experimentally that normal bile would cause acute hemorrhagic necrosis of the pancreas only during the period of active digestion.

While the production of experimental pancreatic necrosis was quite easy, the substances injected into the pancreatic duct were all injected under unknown pressures.

Archibald[7], appreciating that gallstones were not always present to block the outflow of bile in the cases of necrosis, started investigations and found a sphincter muscle at the duodenal papilla, and later found that this sphincter had been described by an Italian physiologist, named Oddi. He then determined to inject various substances under a pressure which the sphincter of Oddi would resist, that is, a pressure of from 400 to 700 mm. of water. Infected ox bile, injected under a pressure of 500 mm. of water, produced extensive haemorrhagic necrosis. Sterilized human bile produced slight necrosis of the parenchymal cells, detected only by the microscope. Sterilized human bile, deprived of its mucin, produced a much more extensive necrosis. These experiments demonstrated that a pressure considerably greater than that at which bile would be excreted, that is, 350 mm. of water, was necessary to produce an effect upon the pancreas. No physiological means of increasing the pressure in the biliary system was found.

---

*Read before the New England Surgical Society, at Worcester, September, 1921.

Judd and Mann[8] took up these experiments at this point and demonstrated that even though the gall-bladder is a muscular organ, contraction of it will not increase the pressure in the biliary system more than 50 mm. of bile. They also demonstrated that by causing the violent muscular effort of retching, the pressure in the biliary system could be increased to 500 and 1000 mm. of bile. They then injected normal bile from the animal operated upon into the biliary system at pressures of 500 and 1000 mm. of bile. The effect was so slight under both pressures that they considered that bile injected into the pancreatic duct under physiological conditions, had no effect upon the pancreas.

In the light of Archibald's and Brocq's experiments, there are several points to be considered in these statements of Judd and Mann. Archibald stated that normal sterile bile produced no macroscopic changes, but that microscopically there was some necrosis when introduced under the conditions used by Judd and Mann. Judd and Mann did not give us the microscopical findings.

Brocq[6] states that normal bile injected into the pancreatic duct would produce haemorrhagic necrosis only if injected during the process of digestion. Archibald also demonstrated that changed bile, that is, either infected bile or bile deprived of its mucin, or bile rich in salts, would produce much more extensive necrosis than normal sterile bile.

If we now combine the experiments of Archibald, Brocq, and Judd and Mann, we have all the steps necessary to produce pancreatic necrosis under physiological conditions. That is, Archibald has denonstrated that the sphincter of Oddi will resist a pressure of from 400 to 600 mm. of water, and that infected bile, or bile with little mucin or an increased proportion of bile salts, introduced into the pancreatic duct under that pressure, will cause pancreatic necrosis. It is fair to assume that in cases of pancreatic necrosis we have an infected or changed bile. Even if we do not have an infected or changed bile, Brocq has demonstrated that normal bile introduced into the pancreatic duct during active digestion, will produce necrosis. Many cases of pancreatic necrosis in humans occur soon after a hearty meal. Judd and Mann furnish the one missing link by demonstrating that the pressure in the biliary system can be increased to 1000 mm. of bile by violent muscular effort, such as retching.

Besides these experiments we have four autopsy reports by Opie[1] which deserve much more serious consideration than has been given to them, for there are few autopsy reports that have gone into details as to the relation of the necrosis of the pancreas to the ducts, and the anatomical relation of the ducts to each other. These four autopsies which gave us these details must therefore be considered carefully. The first is the autopsy report of a case of Eliot. A patient, after long-continued vomiting, had a severe attack of epigastric pain, and died in three days. At autopsy, an acute haemorrhagic necrosis of the pancreas was found. The duct of Wirsung was very small and the duct of Santorini, with a normal opening into the duodenum, was the main duct of the organ. The necrosis was localized almost entirely about this duct.

The second case, that of Bassett, was an acute haemorrhagic necrosis which was localized about the duct of Santorini, which drained only a small part of the head of the gland, and was patent throughout its course into the duodenum.

Two other cases—those of Johnston—were acute haemorrhagic necrosis in which the duct of Santorini was the main duct of the organ, with a normal opening into the duodenum.

There are several facts to be considered in connection with these cases. Brocq, and others, have shown experimentally that duodenal contents injected into the pancreatic duct will produce hemorrhagic necrosis. In all four of these cases, the main duct of the pancreas, or the one about which the necrosis was localized, was the duct of Santorini. In two, the necrosis was definitely limited to the area drained by the duct of Santorini, in one a very small area, and in the other a large part of the gland.

It would seem from the consideration of these reports that we must consider not only retrojection of changed bile and normal bile during the process of digestion, into the pancreatic duct, but also retrojection of duodenal contents into the duct of Santorini, as causes of acute pancreatic necrosis.

Judd and Mann reject Archibald's theory as to the cause of pancreatitis, partly because of the slight effect of normal bile on the pancreas, as stated above, and partly because the proper anatomical relation of the ducts occurs so seldom. That is, the anatomical arrangement of the duct of Wirsung and common duct must be such that a spasm of the sphincter of Oddi or a small stone at the papilla can close the common duct without closing the duct of Wirsung at the same time. Observations made on 170 autopsies, demonstrated this to be possible in only nine out of two hundred individuals. Opie[1] examined one hundred cases at autopsy, and found that the arrangement of the ducts was such that it could occur in thirty out of one hundred. Deaver and Sweet[9] also use this as an argument against Archibald's theory; but if we consider the number of cases of pancreatitis of whatever variety, we would quite certainly not have more than nine cases in each two hundred individuals. If we accept the clinical evidence as given us in Opie's four autopsy reports, that retrojection of the duodenal contents may cause pancreatic necrosis, the anatomical arrangement of the ducts would be such as to permit the occurrence in ten out of each hundred individuals (Opie); that is, the duct of Santorini is the main duct in that proportion of individuals. It would seem, therefore, that we have sufficient experimental and clinical proof, and the proper anatomical relation of the ducts in a sufficient number of individuals, to warrant us in saying that the retrojection of bile or duodenal contents into the pancreatic ducts may cause pancreatic necrosis.

Deaver and Sweet, and Judd and Mann have accepted Maugeret's theory that pancreatitis is caused by an infection of the pancreas from the gall-bladder, through the lymphatics of the gall-bladder to the lymph-nodes of the cystic duct, thence by periductal lymphatics through the glands, along the common duct to the glands at the head and margin of the pancreas, thence by regional lymphatic distribution in the head of the pancreas.

Archibald is opposed to this theory on the ground that it is difficult for an infection to pass from the gall-bladder to the pancreas against the lymphatic current and through so many sets of glands.

Graham[10], and others, have, however, seemed to prove that it is quite possible for infection to pass in this way. One objection which we have to this theory is that there is no experimental proof that an acute haemorrhagic necrosis can be caused by infection through the lymphatics. Graham shows bacteria in the interstitial tissue of the pancreas, but says nothing about pancreatic necrosis.

It is very unsatisfactory to leave the solution of the problem of the etiology of acute pancreatitis here, but it seems impossible to go farther unless we are willing to concede that acute pancreatic necrosis, and pancreatitis so often found in association with infections of the biliary system, are two distinct diseases with different causes.

It is difficult to discuss a subject when so little is known about the pathologic findings, as we know about those of pancreatitis associated with infections of the biliary system. In fact, some pathologists go so far as to say there is no such condition as the chronic pancreatitis which surgeons find so often associated with gallstones. So far as we can learn, the pancreatitis associated with infections of the biliary system is an inflammatory change in the interstitial tissue of the gland, and is so spoken of by Opie. Graham has also shown bacteria in the interstitial tissue of the gland in certain cases of experimental cholecystitis. On the other hand, the condition found in the pancreas in acute pancreatic necrosis, is primarily a necrosis of the parenchymal cells, and in certain cases at least, the necrosis is localized along the pancreatic duct involved, as demonstrated by Opie.

Is it not possible, therefore, that we are dealing with two distinct diseases due to different causes?

It is impossible to read the articles of Deaver, and others, who accept Maugeret's[12] theory as to the cause of pancreatitis, and those of Archibald, and others, who believe in his theory, without feeling that they are discussing two different diseases.

Archibald, and others, produce pancreatic necrosis experimentally almost at will, by the injection of bile and duodenal contents into the pancreatic ducts under physiological conditions, while those who consider that pancreatitis is due to an infection, report no experimental cases of acute haemorrhagic necrosis due to lymphatic infection. Graham shows bacteria in the interstitial tissue of the pancreas, yet says nothing of pancreatic necrosis. Deaver and Sweet[9] state that pancreatitis is a lymphatic infection of the gland, and Graham[10] shows the bacteria present in the interstitial tissue, but no bacteria are found by smear or culture in the cases of pancreatic necrosis. Deaver[9] states that in pancreatitis the gall-bladder or ducts show an infection in 91 per cent., while in pancreatic necrosis it would be difficult to state with accuracy that they were infected in 50 per cent. of the cases.

If pancreatic necrosis is due to the same cause as the pancreatitis found in conjunction with cholecystitis, is it not rather extraordinary that so few cases of pancreatic necrosis occur? Pancreatitis has been found to be present in from 23.8 per cent. to 50 per cent. of the cases of cholelithiasis. Among such a large number, should we not expect more cases of pancreatic necrosis? We have been able to find but 42 cases in the records of the Massachusetts General Hospital, and gallstones were present in but 50 per cent. of those.

In pancreatitis of the interstitial type, the tendency is to recover, no matter what operation is done, while in pancreatic necrosis, death occurs in at least 70 per cent. of the cases.

To sum up the preceding it is my belief that the pancreatitis associated with infections of the biliary tract is an inflammation of the interstitial tissue due to an infection, frequently through the lymphatics of the biliary systems; that acute pancreatic necrosis is a necrosis of the parenchymal cells due to a retrojection of bile into the duct of Wirsung or to a retrojection of duodenal contents into the duct of Santorini.

The interstitial pancreatitis may be acute or chronic, while the cases of pancreatic necrosis are always acute. There are undoubtable cases of recurrent pancreatic necrosis in which only a small area of the gland is involved. This is an aseptic necrosis and is absorbed, leaving little or no evidence of it later. This has been demonstrated experimentally by Whipple[13].

It is also interesting to note that in 43 cases of acute pancreatic necrosis, 22 occurred in patients without gallstones, and of these, 17 had had one or more previous attacks of epigastric pain.

In considering the cases of pancreatic necrosis in this report I have included only those cases which showed definite macroscopic evidence of pancreatic involvement; the great majority of these were cases of acute hemorrhagic necrosis of the pancreas. Acute abscess of the pancreas has been included because it is impossible to separate them from hemorrhagic necrosis which has broken down. The total number of cases is 56, 42 of which were found in the Massachusetts General Hospital records, while 14 were private cases.

As to symptomatology, there is nothing unusual. The pain is a sudden, severe epigastric pain. The patient at once goes into shock of greater or less severity, depending upon the extent of the lesion.

The pain, after the first severe attack, becomes a great distress, not only in the epigastrium but frequently in the back. Patients will frequently say the pain is not severe, but the distress is almost unbearable. They move about in bed and impress one as being in great distress. The pulse is rapid, ranging from 100 to 160, often too rapid and small to count. The temperature, subnormal during the first few hours, gradually rises to 100°, rarely more. The patient is cyanotic, varying with the severity of the condition. Nausea and vomiting are usually persistent. There is leucocytosis, varying between normal and 15,000.

The abdomen contains bloody fluid and is very slightly tender all over, with definite tenderness over the pancreas, starting in the region of the gall-bladder and extending across the abdomen a varying distance, depending upon the extent of the involvement of the pancreas. This tenderness is always present in the first few days if carefully looked for, but may not be so evident after the acute stage passes. The careful localization of this tenderness is frequently the only means of making a differential diagnosis between an acute cholecystitis and a pancreatitis. Two cases have recently been reported by a surgeon, who stated that there was no abdominal tenderness in either case. If this were true after a painstaking examination of the whole abdomen, it would be very unusual.

Another important physical sign, when present, is tenderness at the left costovertebral angle, due to involvement of the tail of the pancreas. Obstipation is a symptom which is always present early, and associated with the pain and nausea, as it usually is, makes the diagnosis of intestinal obstruction a frequent one. In fact, among forty-two cases, a caecostomy was done once and an ileostomy twice. The various tests, such as Cammidge's, have proved of little value.

We must, I feel sure, be much more thorough in our examinations of the pancreas at operation than formerly, for short, sharp attacks of epigastric pain may be due to the pancreas and yet produce very slight evidence in the gland. It is, I believe, quite possible to have small areas of parenchymal necrosis which disappear, leaving practically no trace, as stated by Whipple.

Nicoll[14], of London, has removed sections of the gland in a number of cases of this kind, and has found evidence of necrosis under the microscope which was not visible to the naked eye. Removal of a section of the pancreas in many of these cases would be of great value, but the danger of pancreatic leakage has deterred us from removing the sections. Nicoll, however, reports no bad results.

As to treatment, we must be guided by what we believe to be the etiology of the type of disease we have to treat. If we accept Maugeret's theory we must remove the gall-bladder, the source of the infection. If we accept Graham's experiments, which demonstrate the infection of the gall-bladder from a hepatitis through the lymphatics, we must, I think, admit the lymphatics of the hepatic and common ducts may become infected from the liver, and the pancreas infected through them. If this is a fact, removal of the gall-bladder would be of little value. However, removal of the gall-bladder is the only rational treatment under the circumstances, as we can do no more. If we accept Archibald's theory that acute pancreatitis is due to retrojection of bile into the pancreatic duct because of obstruction at the papilla of Vater either by a spasm or a stone, something must be done to relieve the pressure in the ducts, that is, a cholecystostomy or a choledochostomy, is the rational operation. Archibald suggests drainage from three weeks to several months, or section of the sphincter of Oddi at the papilla, which at once reduces the possible pressure in the ducts to 60 or 70 mm. of water. Drainage of the biliary system for three weeks appears to be rational, as it gives the pancreas an opportunity to return to normal and permits any temporary condition which may have caused a spasm of the sphincter of Oddi to subside, but it is difficult to see what advantage longer drainage can be. It is not likely that drainage for a longer time can make a permaent change in any condition which has caused the spasm of the sphincter of Oddi. If there is hyperacidity which has caused the spasm, the only rational treatment is section of the sphincter, suggested by Archibald, and treatment of the hyperacidity. But we are discussing the treatment of acute pancreatitis, and in the cases of acute hemorrhagic necrosis of the pancreas, the condition of the patient does not often permit any prolonged operative procedure. In such cases, the simplest method of drainage of the biliary system should be carried out, that is, cholecystostomy.

Archibald advises against any drainage of the capsule of the pancreas. He says that the shock associated with the disease is due to the products of digestion, and drainage would be of no value. We are opposed to this theory on the ground that many of these patients improve at once after drainage of the fatty capsule of the pancreas, or of the pancreas itself.

If we believe that retrojection of duodenal contents into the duct of Santorini may cause pancreatitis we have no method of drainage of the duct that will be effective; we must, therefore, depend upon drainage of the fatty capsule of the pancreas, or the pancreas itself.

Pierre Delbet[15] suggests section of the common duct and a union of the gall-bladder with the small intestine. This would obviate any possibility of retrojection of bile into the pancreatic duct, but it is a procedure which very few of the cases of acute pancreatic necrosis could withstand.

The results of the various methods employed in 56 cases of acute pancreatic necrosis, collected from the records of the Massachusetts General Hospital and my own records, are as follows:

Total number of cases	56
Total mortality	60.7%
Mortality of pancreatic necrosis without the cases of abscess	72.3%

OPERATION.	TOTAL NO.	SEVERE.	MILD.	DEATHS.	MORTALITY.
No Operation	6	6		6	100%
No Drainage or Drainage of Abd. Cavity	14	10	4	12	86%
Drainage of Pancreas including Abscesses	29	25	4	8	28%
Drainage of Pancreas without Abscess	20	18	2	11	55%
Drainage of Abscess alone	9	7	2	0	00%
Drainage of Biliary System	3	2	1	2	66%
Drainage of Biliary System and Pancreas	3	2	1	3	100%

There are six cases in which the biliary system was drained; in three the fatty capsule of the pancreas was also drained. The mortality of the group was 83 per cent. There were 20 cases of acute haemorrhagic necrosis of the pancreas in which the pancreas or fatty capsule of the pancreas was drained without any other procedure, and the mortality was 55 per cent.

While this group of cases is small it seems to give some indication as to what should be done, and indicates that drainage of the fatty capsule of the pancreas, or the pancreas, gives better results than any other procedure. Drainage of the gall-bladder or, better still, the common duct if the condition of the patient will permit, may be and, at least theoretically, would be a valuable addition to drainage of the fatty capsule of the pancreas, or pancreas itself.

Archibald advises waiting on these patients until they have recovered from the shock, as he feels it is due to the absorption of split proteins, and operation will do no good. While this may be correct, theoretically, we are convinced, after operating upon a number of these cases, that operation the moment the patient's condition will permit, with novocain and gas oxygen anesthesia, will give far better results than waiting. The ideal operation would be drainage of the common duct, drainage of the fatty capsule of the pancreas, and pancreas itself. If the common duct cannot be drained easily, a cholecystostomy should be done instead, and if only little can be done, drainage of the fatty capsule of the pancreas alone should be done.

CONCLUSIONS.

1. Experimental and clinical findings lead us to believe that there are probably two types of acute pancreatitis, with quite different etiology.
   a. Interstitial pancreatitis due to infection of the interstitial tissue, the infection coming frequently from the biliary system.
   b. Pancreatic necrosis, a necrosis of the parenchymal cells, due to retrojection of bile into the duct of Wirsung or duodenal contents into the duct of Santorini.
2. The treatment of the interstitial type, due to infection from a cholecystitis, is cholecystectomy.
3. The treatment of pancreatic necrosis is drainage of the fatty capsule of the pancreas, and the pancreas itself. In addition, if the condition of the patient will permit, either the sphincter of Oddi may be cut, a choledochostomy or cholecystostomy done, depending upon the condition of the patient and the condition of the gall-bladder and ducts found at operation.

REFERENCES.

[1] Opie: Diseases of the Pancreas, 1910, 2nd edit.
[2] Flexner: Johns Hopkins Hosp. Reports, 1900, vol. 9, p. 743.
[3] Hlava: Bull. Internat. de l' Acad. des Sc. de Bohème, 1890, vol. 4, p. 139.
[4] Gulcke: Ergebn. d. Chir. u. Orthop., 1921, vol. 4.
[5] Brocq: Compt. Rend. Soc. de Biolog., 1919, vol. 82, pp. 371 and 510.
[6] Brocq (reported by Delbet): Bull. et Mem. de la Soc. de Chir. de Paris, 1914, vol. 40, p. 24.
[7] Surg. Gyn. & Obs., 1919, vol. 28, p. 529.
[8] Judd and Mann: Jour. A. M. A., July 16, 1921.
[9] Deaver and Sweet: Jour. A. M. A., July 16, 1921.
[10] Graham: Arch. of Surg., Jan., 1922.

[11] Graham: Arch. of Surg., Jan., 1921.
[12] Maugeret: Recherches que la pathogenie des pancreatites infectionere.
[13] Whipple and Goodpasture: Surg. Gyn. & Obs., 1913, vol. 17, p. 591.
[14] Nicoll: Brit. Med. Jour., 1919, vol. 2, p. 625.
[15] Pierre Delbet: Bull. et Mem. de la Soc. de Chir. de Paris, 1914, p. 24.

[The discussion of Dr. Jones's paper appeared in the JOURNAL of February 16, 1922, No. 7, Vol. 186, pages 213-216, after the other papers of this series.]

Original Articles.

THE INDICATIONS FOR OPERATION IN THE TREATMENT OF INJURIES INVOLVING THE BRAIN.

By Donald Munro, M.D., Boston,

Assistant Out-Patient Surgeon, Boston City Hospital.

The indications for operation in the treatment of injuries involving the brain are three:

First, a compounded fracture of the skull with injury to the brain.

Second, a depressed fracture of the skull involving either both tables or the inner table alone.

Third, and most important of all, an increase in the intracranial pressure.

Exclusive of the more or less specialized forms of fracture of the skull—the compound and the depressed varieties, I do not consider fracture *per se* an indication for operative interference.

Not infrequently the first thought and all of the attention of a physician who sees a patient who has received an injury to his head is devoted to determining whether or not there is present a fracture of the bony covering of the brain.

I wish to show that this is, except in the case of a compound or depressed fracture, a mistake that may cost the patient his life, or at least decrease his chances of recovery, and furthermore, that having obtained this information, the physician is no better off from the point of view of locating the injury than if he had made no effort in this direction at all.

Patients who have received an injury to the brain do not develop symptoms and do not die from the fracture which may or may not be present in the bones of the head. They do develop symptoms, and they do die as a result of the increase in the intracranial pressure which accompanies the fracture, or which not infrequently is present in the absence of any fracture.

COMPOUND FRACTURE.

The indications for operation in compounded fracture of the skull are clear. Just as in a compounded fracture of any other bone we have at the very least a potentially infected wound, and steps must be taken to prevent this infection from spreading to surrounding clean tissues—in this case, of course, primarily the brain and meninges. Briefly, this may be best accomplished in the majority of cases by the process known as debridement, developed and used in the medical services of the allied armies during the war.

DEPRESSED FRACTURE.

In a depressed fracture of the skull it is important to remember that there may be a depression of the inner table of the skull alone, without a break in the contour of the outer table. Consequently, it is at once apparent that in these cases the X-ray examination of the skull becomes of greatest importance. I believe that more information may be obtained with less effort if stereoscopic plates in both anterior-posterior, and lateral directions are taken at once if possible. Of course, many times, with excited or restless patients this is impossible, but I believe that this should be the aim of the roentgenologist. These depressions should be either raised or, if that is impossible, removed.

This, then, briefly, covers the cases of injury to the skull and its contents in which the fracture in itself has any bearing on the operative treatment of the case.

INCREASED INTRACRANIAL PRESSURE.

Increased intracranial pressure as an indication for operation in the treatment of injuries of the brain is of paramount importance. The determination of its presence or absence should be the first information to be ascertained in any such case. Neglect of this all important information in the diagnosis, and a cloudy or mistaken conception of its relation to the future treatment and the present condition of these cases, I believe to be the one great factor that as late as 1916 kept the death rate in this class of case as high as 50 per cent.[1]

To appreciate the importance of a pathological increase in intracranial pressure, it is necessary to have a knowledge of some of the more important points of the anatomy and physiology of the cerebrospinal fluid, its origin, distribution, and ultimate destination.

PRODUCTION AND DISTRIBUTION OF THE CEREBROSPINAL FLUID.

Magendie,[2] whose extensive researches in this subject were published between 1825 and 1842, was the first to appreciate the great physiological importance of this fluid. Through his investigations it was shown that not only are the ventricles and subarachnoid space normally filled with fluid, but that the fluid passes freely from one to the other of these divisions. Since that time many workers have added to our information in this field through chemical, intra-vitam staining, and experimental methods culminating finally in a series of experiments conducted recently by

Dandy[3] at Johns Hopkins, and published in 1919 and previous years. It is not necessary to repeat here a detailed description of this work, but certain points should be emphasized. The cerebrospinal fluid is secreted by the choroid plexus practically in its entirety.[2, 3] This plexus lies in the two lateral, the third and the fourth ventricles. From the lateral ventricles the fluid passes by way of the foramina of Monro into the third ventricle and receives its quota from the choroid plexus there. The fluid then passes through the acqueduct of Sylvius into the fourth ventricle and receives its quota from this portion of the plexus. From the fourth ventricle the fluid passes by way of the foramina of Magendie and Luschka to the cisterna magna, which in turn communicates with the entire cerebral and spinal subarachnoid space. Approximately one-half of the total quantity of cerebrospinal fluid is contained within the spinal subarachnoid space. The ventricles contain from 20 to 30 c.c.[4]

ABSORPTION OF THE CEREBROSPINAL FLUID.

This then is the method of production and distribution of the cerebrospinal fluid. We must now consider its absorption. In connection with this it is well to recall that the subarachnoid space lies beneath the arachnoid and above the pia, that this space closely invests every part of the central nervous system, and, owing to its relation to the pia, comes into intimate contact with the surface capillaries and many of the larger vessels of the entire brain and cord. From this great space the cerebrospinal fluid passes into the venous circulation either by way of a filtration process *via* the arachnoidal villi, or by diffusion into the capillaries of the subarachnoid space. The investigations of Key and Retzius, Renier and Schnitzler, Sicard and Cestan, Frazier and Peet, Mott, Dandy, and Blackfan, and others have definitely shown that the cerebrospinal fluid reaches the venous circulation, and that the lymphatic absorption, if any, is so slow and of such a small amount that it can be disregarded.[5] The only point in question at this time is just what the channels are that the fluid passes through after leaving the subarachnoid space, and before reaching the venous circulation.

In brief then it can be said that the cerebrospinal fluid is secreted by the choroid plexus in the ventricles; passes from them by way of the foramina of Magendie and Luschka into the cisterna magna and thence is distributed over the entire surface of the central nervous system within the subarachnoid space, and in intimate relation with the pial vessels whence it is absorbed into the venous circulation.

INTRACRANIAL PRESSURE.

Normally the secretion of the cerebrospinal fluid is constant.[6] Necessarily, therefore, the absorption must also be constant. As a corollary to these two facts the secretion must be carried on at a sufficient rate to maintain pressure enough to promote absorption. This secretory pressure depends on two variable factors—first, the rate of secretion, and second, the rate of absorption. With a variation in either the rate of secretion or the rate of absorption, there will be a direct and resultant variation in the pressure. Normally this pressure, to which we can now give the name of intracranial pressure, varies with and is equal to the pressure in the venous sinuses.[7] It depends upon the amount of cerebrospinal fluid present, and also upon the systemic blood pressure and respiration.[8] Interference with the arterial circulation of the brain causes only a transitory lowering of the intracranial pressure, at once followed by a return to normal as would be expected. On the other hand, equally as would be expected, interference with the venous circulation, by impeding the absorption of the cerebrospinal fluid causes an immediate and more or less constant increase in the intracranial pressure.[9] In view of the distribution of the cerebrospinal fluid it is apparent that any increase of intracranial pressure will have an effect upon the entire central nervous system, and more especially upon the cerebrum and medulla. That this must be so is clear when we recall that not only is this pressure exerted upon the more superficial aspect of the cerebrum and medulla through the subarachnoid space and cisterna magna, but also is exerted in a similar manner and to an equal extent upon the innermost surfaces of the cerebrum and medulla by way of the ventricles. The cerebrum and medulla, therefore, may be said to be subjected to a process of squeezing whenever the intracranial pressure is raised. The amount of squeezing and consequently the amount of damage to the delicate nervous tissue of the brain depends upon the amount of the increase in the intracranial pressure.

Here, then, is a possible source of injury to the brain and medulla the extent of which depends only upon the height to which the intracranial pressure rises. That this may cause the death of the individual subjected to this rise is obvious, especially when we take into consideration the fact that regulatory centres, upon the normal functioning of which the continuance of life depends, are located in the medulla. Furthermore, it is evident that any lesion which interferes with the normal cerebral venous circulation will give rise to this increase of pressure. Enumeration of the possible lesions that may cause this shows on the one hand how unimportant a relation a linear fracture of the bony coverings of the brain bears to this serious condition, and on the other hand how important and what a direct effect upon life such conditions[10] as asphyxia, reflex disturbances of vasomotor control leading to acute congestion and oedema of the meninges, effusion of blood into the ventricles or between the meninges, or in the substance of the cord or brain, contusion and laceration of the brain by contra-coup or direct violence, hemorrhage into the subarachnoid space, and other similar conditions may have. Treatment must be directed toward relieving this pressure. This may be done in one of two ways: first, by withdrawing some of the cerebrospinal fluid, and second, by allowing the brain more room to expand, or in other words by a decompressive operation upon the skull.

MEASUREMENT OF CEREBROSPINAL PRESSURE.

The ability to diagnose correctly this important and dangerous condition depends upon our ability to measure, first, the normal cerebrospinal pressure, and second, an increase in cerebrospinal pressure.

The normal cerebrospinal pressure has now been measured many times, and a variety of manometers have been devised for this purpose. The pressures recorded in the case reports below were measured by the mercury manometer devised by L. H. Landon while at Frazier's clinic in Philadelphia. Briefly, it consists of a U tube containing mercury, against one arm of which is set a millimeter scale for convenience in measuring the rise of the mercury columns. With the adult patient lying quietly on his left side with the entire spinal column horizontal and the top of the columns of mercury on a level with the needle, which is inserted as for an ordinary lumbar puncture, the normal pressure varies between 6 and 10 mm., and in the large majority of cases is in the immediate neighborhood of 8 mm.[11, 20] New-born babies tend to have a somewhat lower reading than adults, but otherwise show no great variation. Any reading above 10 mm. Hg. is abnormal and is evidence of an increase in intracranial pressure.

OTHER SIGNS OF INCREASED CEREBROSPINAL FLUID PRESSURE.

Cases which are suffering from an injury to the brain, and in whom can now be demonstrated, by means of the spinal manometer, a definite rise in intracranial tension or pressure, also show certain more or less constant changes in the eye grounds, blood pressure, pulse rate and volume, temperature, respiratory rate, and reflex activities. These changes were previously considered to be indications which controlled absolutely the treatment of the case at any given time. They are still considered so in many clinics, and by some neurological surgeons are considered of more importance than the measurement by manometer of the intracranial pressure. I believe this to be a mistake, and within the last two months have seen one case with a rising pulse, normal blood pressure, and essentially normal reflexes, who showed at lumbar puncture a pressure of 30 mm. Hg., and at operation a large middle meningeal clot, and have known of another case in which a demonstrated pressure of 40 mm. Hg. was ignored and autopsy revealed a large subdural hemorrhage.

EYE GROUNDS.

Of all of these concomitant signs of pressure that of the eye grounds is the most constant and by far the most valuable. A small rise of pressure above the normal may dilate the veins of the retina, and cloud the nasal margin of the disc. A further rise will produce the classical choking of the disc.[12]

PULSE, BLOOD PRESSURE, ETC.

The changes in pulse, blood pressure, respiration and temperature, are themselves due to the rise in intracranial pressure, and indicate nothing more than successive stages in the increase in this pressure.[1, 3] They are not directly connected with the injury and have no bearing upon the diagnosis, treatment, or prognosis of the case other than that indicating further pressure.

Given a case which has been struck sufficiently hard upon the head to produce symptoms, the least severe injury to the contents of the skull that could occur would be an irritation of the meninges. Injuries of greater magnitude would, of course, include laceration and contusion of the brain of varying extent, and massive intra- or extra-dural hemorrhages. In connection with this,[14] Kolmer, writing in Frazier's "Surgery of the Spine and Spinal Cord," states that "as the result of acute inflammatory changes in the meninges, with great increase in the volume of fluid, this regulating mechanism (controlling the intracranial pressure and volume) is disturbed." In other words, more cerebrospinal fluid is secreted than is absorbed, and as a result the intracranial pressure rises. With an extensive injury to the brain surface itself, or with a large intra or extra-dural hemorrhage, we have also in addition to the meningeal irritation an actual blocking of a certain portion of the subarachnoid space. That is, a blocking of a part of the absorptive area. As a result of this we have an actual excess of cerebrospinal fluid. The brain being non-compressible and this fluid having no place to go stays within the residual subarachnoid space under a greater pressure. This pressure in its turn causes a more or less local cerebral anæmia and resulting cerebral venous stasis. This condition is spoken of by Sharpe[15, 16] as the First Stage of Compression. The symptoms are headache, drowsiness, stupor, or possibly marked excitement. The retinal veins may be engorged. The manometric cerebrospinal fluid pressure is from 12 to 14 mm. Hg. Repeated lumbar puncture with withdrawal of sufficient cerebrospinal fluid to lower the pressure to normal will frequently cure patients in this condition.

The following will serve to illustrate the type of case referred to in the preceding paragraph:

J. A. W.—Male, 40, white, married; working as freight conductor. Seen May 10, 1921, in consultation with Dr. A. P. Lowell, Fitchburg, Mass. *Diagnosis:* Multiple Contusions and Abra-

sions, Contusion and Oedema of the Brain—probably contra-coup—with Subarachnoid Hemorrhage, Severe Concussion. *Treatment:* Cerebrospinal fluid drainage by lumbar puncture. ***Discharged:*** Cured. The patient was found unconscious on the floor of a caboose at 2.30 A. M., May 8, 1921. No history was available. Carried at once to Burbank Hospital, Fitchburg. On admission he had two generalized convulsions and was in considerable surgical shock with pulse about 120. Was also noisy, irrational, and very restless. May 9, 1921, condition improved. No further convulsions and patient quieter. His pulse dropped to 72, and he recognized his wife. This A. M. (May 10, 1921) pulse still dropping —around 44. Blood pressure 120. Seen at 3 P. M. when he was conscious and rational, but somewhat stuporous. Had a complete amnesia dating from at least one week previous to the accident and continuing to the present. Recognized his wife and sister, but did not know the day of week nor the month. Examination showed lacerated wounds of scalp and outer canthus of left eye and multiple contusions and abrasions over the body. Examination of the *heart, lungs, abdomen,* and *bony skeleton* is normal everywhere. *Urine* negative. *Neurolgical examination* shows no changes in either the *somatic* or *splanchnic motor* or *sensory* systems. The *cranial nerves* are all normal except for complete deafness in the left ear. Eye grounds are normal. There is no *ataxia, adiadokokenesis* nor *astereognosis.* The speech is thick, but otherwise not changed. Mentally he is dull and apathetic, but answers questions correctly except as above. There is a complete *amnesia* dating to one week previous to the accident and extending up to the present. *X-rays* of the skull show no fracture and no evidence of any intracranial pressure. *Lumbar puncture* showed a pressure of 14 mm. Hg. Sufficient fluid was withdrawn to reduce this pressure to 7 mm. Hg. without symptoms. The cerebrospinal fluid was colored and mixed with blood which evidently came from a subarachnoid hemorrhage higher up. Last seen on June 3, 1921, having been out of the hospital since May 29. On this former date had no symptoms of any sort and is to go back to work in a day or so.

G. W.—Male, 18, white, single; working as a lineman for an electrical power company. Seen May 16, 1921, in consultation with Dr. H. W. Ellam of Gardner, Mass. *Diagnosis:* Contusions of Neck, Shoulders, and Back—Lacerated Wound of Posterior Part of Scalp—Fracture of the Right Occipital and Parietal Bones—Subarachnoid Hemorrhage probably due to contusion of Cerebellum—Moderate Intracranial Hypertension. *Treatment:* Cerebrospinal fluid drainage by lumbar puncture. *Discharged:* Cured. May 15, 1921, while at work was struck on back of head, neck and shoulders by an "insulator" said to have weighed about fifty pounds. Taken at once to the hospital following the injury. No loss of consciousness or memory at any time. Said to have been very talkative and excited immediately following the accident. On entering hospital temperature was 101 and pulse 120. A few hours after entrance became stuporous and vomited repeatedly, being unable to take any food. Today is somewhat better, but still unable to take food and complaining of nausea and dizziness on sitting up. Temperature now normal and pulse 72. *Physical examination* of *heart, lungs, abdomen,* and *bony skeleton* normal except for contusion of shoulders and back, and a lacerated wound of posterior portion of scalp. *Blood pressure* 130/70. *Urine* negative. *Neurological examination* shows no changes in either the *somatic* or *splanchnic motor* or *sensory* systems. *Cranial nerves* are all normal except for a moderate nystagmus of both eyes with the quick component toward the right, on extreme rotation of the eyes to the right. *Eye grounds* normal. There is a slight past-pointing toward the right with the right hand in the finger to finger and finger to nose tests. The left is normal. There is no *adiadokokenesis* nor *astereognosis. Mentally* he is alert and rational. No changes in his speech. *X-rays* of skull show a linear fracture of both tables of the skull about 1½ inches long running at right angles to and crossing the occipito-parietal suture at the junction of the lower and middle thirds on the right. *Lumbar Puncture* showed a pressure of 14 mm. Hg. About 5 cc. of cerebrospinal fluid was withdrawn which lowered the pressure to 6 mm. Hg. without producing any symptoms. This fluid was colored by blood from a subarachnoid hemorrhage higher up. When last heard from in November, 1921, this patient had been back at work and entirely without symptoms since early in June.

It is to be noted that in neither of these cases were there any reflex changes, nor other objective symptoms of any characteristic degree. One suffered from a severe concussion, and the other did not. One had a fracture of the skull, and the other did not. In both, however, the cerebrospinal fluid pressure was raised to an equal degree. There was a subarachnoid hemorrhage, and withdrawal of enough cerebrospinal fluid to reduce the pressure to normal effected a prompt cure in both cases.

The next stage in increasing intracranial pressure is spoken of by Sharpe[17, 18] as the Ideal Operative Stage. In this stage the rising intracranial pressure causes a more extensive and complete cerebral anæmia. If the pressure is due to an extradural lesion, such for example as a middle meningeal hemorrhage, or a depressed fracture, the anæmia is more localized and we have symptoms due to the impairment of the particular part of the cortex underlying the lesion. Owing to the falx and the tentorium the majority of the pressure exerted by the extradural lesions is in this stage localized to one hemisphere, and the symptoms are not, therefore, as grave, due to the fact that the medulla is protected. With an intradural lesion, however, the protection afforded by the falx and tentorium is of not so much avail, and consequently the medulla is included in the more or less generalized cerebral anæmia. The first effect of this anæmia on the medulla is said to stimulate the vagal nucleus, and does in any event produce a slowed pulse, and also a slight rise of the peripheral blood pressure due to stimulation of the vasomotor centres. The cerebral venous stasis is more pronounced, and the patient presents symptoms similar to those noted above, only more severe. Manometric readings of cerebrospinal fluid pressure are from 18 to 30 mm. Hg. Treatment consists of subtemporal decompression. Neither at this nor at any subsequent stage of increasing intracranial pressure should more than 1 cc. of cerebrospinal fluid be withdrawn by lumbar puncture for examination. To break this rule subjects the patient to the danger of a herniation of the medulla through the foramen magnum with resulting death.[19, 21]

A. S.—Male, 39, white, married, proprietor of a grain store. Seen April 30, 1921, in consultation with Dr. Thomas Donovan, Fitchburg, Mass. *Diagnosis:* Linear Fracture of Left Temporal Bone—Rupture of Left Middle Meningeal Artery with Extradural Hemorrhage—Medullary Anæmia with marked Intracranial Hypertension. *Treatment:* Left Subtemporal Decompression. *Discharged:* Relieved. April 26, 1921, while moving a 250 pound bag of grain, a pile of similar bags fell over on top of him burying him beneath them. Taken at once to the hospital unconscious. On arrival there became conscious again, and then for a period became unconscious again following which he again became conscious but very stuporous. Speech has been thick at all times, and the stupor has been gradually increasing. Pulse has been constantly around 44, and temperature slightly elevated. Has complained bitterly of a severe frontal headache. April 30, 1921, patient became a little clearer mentally though still complaining of his headache, and still showing the same alteration in his speech. The pulse is now irregular, varying every few hours from 72 to 40. Temperature has risen to 100°. Respirations are normal in rate and character. *Physical examination* of *heart, lungs, abdomen* and *bony skeleton* is all normal. *Urine* normal. *Neurological examination* shows no changes in the *somatic* or *splanchnic motor* or *sensory systems* except for an ankle clonus on the right. *Cranial nerves* all normal. Eye grounds could not be successfully examined on account of the patient's restless condition. Apparently no ataxia. *Diadokokenesis* and *stereognosis* not tested. Mentally very stuporous and dull and difficult to arouse. Answers questions and obeys directions with only a moderate amount of success. *Speech* is very thick and slow and at times the patient is at a loss for a word that he obviously knows and wishes to use but cannot. At these times he will substitute some other word sometimes synonymous and sometimes not. No attempt was made to have the patient read or write. He could not repeat a test sentence and evidently did not understand its meaning. Remembers nothing relating to the accident previous to his first night in the hospital. *X-rays:* thought previous to operation to be negative, but after the fracture had been located at operation it was possible, with the aid of a magnifying glass, to make out a line of fracture previously missed about ½ inch in length in the squamous portion of the left temporal bone. *Lumbar Puncture* showed a pressure of 30 mm. Hg. No fluid was withdrawn. Operation: Typical left subtemporal decompression through a muscle-splitting incision. Bony opening 3 inches in diameter extending from the level of the floor of the middle fossa of the skull. There was a large extra-dural clot evidently from a rupture of the middle meningeal artery although no bleeding point was found. As much of this clot as could be was removed. The dura and arachnoid were then opened and a moderate amount of cerebrospinal fluid under pressure was allowed to escape. Following this the pulse rose from 72 to 120, and the brain which had not been pulsating began to pulsate. The cortex was flattened and injected, but otherwise normal. Dura was closed with a rubber tissue drain between it and the brain on the floor of the middle fossa and the rest of the wound sutured with drainage through the upper end to the dura. The brain was pulsating normally with no sign of herniation at the end of the operation. May 5, 1921, patient now normal in every way. The decompression is entirely beneath the temporal muscle and well protected. All drainage is out. Last heard about in August, 1921, at which time he was back at work free from symptoms.

Should the intracranial pressure still rise, we have a stage in which the symptoms are those of compression of the medulla.[22, 23] The anemia of the medulla becoming more profound, this lack of blood acts as a stimulus to the vasomotor centre which in its turn raises the general arterial blood pressure, and slows the pulse, thus forcing more blood into the medulla and temporarily overcoming the anæmia until the intracranial pressure rises a little further and the process is repeated. In addition to this we have a gradual slowing of the respirations with longer and longer periods of apnea, finally becoming the typical Cheyne-Stokes type of respiration, probably resulting from anæmia of the respiratory centre. It is at this stage that we find the typical choked disc due to an oedema of the optic nerve. The symptoms of this stage then are rising blood pressure with dropping pulse of a full bounding character, and Cheyne-Stokes respiration. The patient is unconscious, and often all reflex activity is abolished. Manometric readings are always above 20 mm. Hg., and may reach

40 or even higher. Subtemporal decompression is imperative at this stage, and even in spite of this operation the patient may die. No patient should be allowed to reach this extreme stage of compression, and were the cerebrospinal pressure measured when the patient is first seen—provided he has recovered from the initial shock of the injury—no patient would reach this stage.

N. P.—White, male, 50, married, born in Greece; works in restaurant. Seen May 24, 1921, in consultation with Dr. R. F. Burns, Fitchburg, Mass. *Diagnosis:* Laceration and Contusion of Right Parietal and Temporal Lobes of Cerebrum,—Medullary Compression with extreme Intracranial Hypertension,—Fracture of Right Occipital and Parietal Bones extending into the petrous portion of the right Temporal Bone. *Treatment:* Right Subtemporal Decompression. *Discharged:* Relieved. Patient speaks no English, having been in this country only a few months and history was incomplete and more or less inaccurate. It is said that on May 19, 1921, patient fell down stairs striking his head against a barrel of cement. Was conscious following this, and able to ride to the hospital in an automobile. Was somewhat dazed and stuporous on admission, but this has cleared gradually up to May 23 on which day he tried to get out of bed, and appeared to be fairly bright. May 24 is not so well, and is again somewhat irrational and stuporous. The temperature has been always more or less elevated up to 101. Pulse has recently been gradually but continuously dropping. May 24 pulse was 65 and respirations were taking on the Cheyne-Stokes character. Physical examination of *heart, lungs, abdomen,* and *bony syeleton* is all normal. *Blood Pressure* is 190/100. *Urine* shows a massive coagulation of albumin with fine and coarse granular casts but no blood. Neurological examination showed the following: *Somatic Motor*—both arms and the right side of the abdomen and right leg are normal in every way. The left abdominal reflexes and both cremaster reflexes are all absent. There is a flaccid paresis of most of the muscles of the left upper and lower leg involving especially the extensors of the thigh, the flexors and tibialis groups of the lower leg. The knee and ankle jerks are present and normal. Stroking the sole of the foot, squeezing of the calf or pressure over the inner border of the tibia elicits reflex motions affecting the toes of the right foot which fan with a plantar flexion of the great toe. Attempts at voluntary motion of the paralyzed leg show as corresponding motions in the other leg. *Somatic Sensory:* there is a diminution, and very probably a complete anesthesia, of the entire left lower leg from the knee down. There are no other sensory changes. *Splanchnic Sensory* and *Motor Systems* are normal. *Cranial Nerves:* examination of these nerves was unsatisfactory and incomplete owing to the patient's inability to co-operate, but the following definite changes were made out: The eye-grounds showed a marked dilatation of the retinal veins, but no hemorrhage, and a definite choking of both discs. The first, eighth, elventh, and the muscular portion of the fifth could not be examined. The other cranial nerves were all normal. *Diadokokenesis* and *Stereognosis* could not be tested. There was no bleeding from the nose, mouth, or ears, and no hemorrhage into either orbit. *Mentally* is very dull and frightened, and objects to having his right eye forcibly opened. *X-ray* shows a horizontal line of fracture, about 3 inches long running forward from the right occipital bone through the parietal, to end in the petrous portion of the temporal bone on this side. This is a linear fracture involving both tables, and is everywhere widely separated. *Lumbar puncture* showed a pressure of 28 mm. Hg. No fluid was withdrawn. *Operation:* Typical Right Subtemporal Decompression through a muscle splitting incision. Bony opening included all of the temporal bone beneath the muscle. Dura was not pulsating, dark blue in color, and very tense. Beneath the dura the brain was found moderately contused and lacerated and covered with old clotted blood. The cortex was flattened and injected, and the pial veins were enlarged in many instances to the size of a normal external jugular vein. There was practically no cerebrospinal fluid in the subarachnoid space exposed at operation. Attempts to puncture the lateral ventricle through the temporal lobe and the third ventricle through the corpus callosum were not successful. There was a moderate herniation of the brain, but no splitting of the cortex. Wound was closed in layers with rubber tissue drainage to the floor of the middle fossa between the dura and the brain. The patient left the table in some shock, but soon rallied, and had an uneventful convalescence. October 11, 1921, comes for examination. Complains of slight dizziness on stooping, but otherwise O. K. *Examination* shows a distinct muscular weakness of the entire left side of body with a diminution of pain and tactile sensation also in this area. There is no paresis or paralysis nor anesthesia, however, and patient walks about all right. There is marked *adiadokokenesis* of the left arm. The *cranial nerves* are all normal, and the eye grounds show nothing but a slight blurring of the nasal sides of the discs. The *decompression* pulsates normally, is not adherent to the skin, and lies entirely beneath the temporal muscle. Fills but does not bulge on stooping. To do light work in a week. *Blood Pressure* 199/125. Last heard about in December, 1921, at which time he was back at work with no complaints.

M. A.—Female, white, 4½ years. Seen July 18, 1921. *Diagnosis:* Extensive fracture of Occiput—Extreme Compression of the Medulla verging on Oedema, with extreme Intracranial Hypertension—Laceration and Contusion of the Brain. *Treatment:* Right Subtemporal Decompression. *Discharged:* Dead. Patient said to have been struck by automobile while playing on the street, on July 16, 1921. Carried home unconscious, where she remained until admitted to the hospital July 18, 1921. Vomited considerably during this time. Seen on day of injury by a local doctor (unknown) who is said to have given no treatment, but to have endeavored to have X-ray plates made of skull. He was not successful in this until the afternoon of July 18, following which the roentgenologist, recognizing the gravity of the case sent the child to the hospital, where she was seen late the evening of the 18th. On admission the pulse was 110, temperature 102 4/10, and the blood pressure 110/70. Some hours previous to being seen, 10 cc. of cerebrospinal fluid had been removed by lumbar puncture, and this fluid was colored with blood from a subarachnoid hemorrhage. *Physical examination of heart, lungs, abdomen,* and *bony skeleton* is normal. There are multiple contusions over the body. The *pulse* is around 110, *temperature* elevated, and *respirations* distinctly slowed and beginning to show a Cheyne-Stokes character. The entire *scalp* is very boggy. *Blood Pressure,* 110/70. *Urine* normal. *Neurological examination* as follows: *Somatic Motor System*: all arm reflexes, abdominal reflexes, and ankle jerks are absent on both sides. The knee jerks are present and equal. The left arm shows a complete flaccid paralysis. *Somatic Sensory* system could not be tested on account of patient's condition. *Splanchnic Motor* and *Sensory* systems: normal except that the left pupil is widely dilated and fixed. The right reacts promptly to light. *Cranial nerves:* The examination was incomplete on account of patient's condition. There was a definite paralysis of the right sixth. Both facial nerves appeared to be normal. There was a marked choking of the left disc with dilated retinal veins, but no hemorrhages. The right disc could not be seen. *Mentally* the patient was unconscious and irrational, very restless, tossing all over the bed, and requiring constant watching, and restraint to keep her from throwing herself out of the bed on to the floor. She was noisy and cried and moaned constantly. *Lumbar puncture* showed a cerebrospinal fluid pressure of 40 mm. Hg. No fluid was withdrawn. *X-ray* not seen, but reported to have shown a linear fracture of the occipital bones, extending from above one ear and running around the back of the head to terminate beneath the opposite ear. *Operation:* Typical Right Subtemporal Decompression under local anesthesia. Muscle splitting incision and bony opening which included the entire squamous part of the temporal bone. On exposure of the dura it was found to be not pulsating, dark colored, and extremely tense. On wide incision considerable dark colored old fluid blood and clots escaped. The brain still did not begin to pulsate although there was no herniation and a successful attempt was made to enter the lateral ventricle with a blunt needle. Considerable bloody cerebrospinal fluid was drained off in this way, slowly, following which the brain volume diminished, and the pulsations returned. Previous to this the cortex was flattened, injected, and the pial veins much enlarged. The portion of the brain exposed at operation had not been lacerated, and there was practically no cerebrospinal fluid in this portion of the subarachnoid space. Following the drainage of the ventricle the patient's pulse rose rapidly, became of poor quality and before the scalp could be entirely closed she died.

The first of these two cases exemplifies a severe type of brain injury treated late in the disease. The patient is again a useful member of society, and at present shows only a relatively small amount of permanent damage considering the severity of the injury, and the complicating diseases from which he suffered.

The second case is inserted as a contrast to the first, and because it exemplifies practically all of the mistakes in treatment that could possibly have been made in this type of injury. First: About sixty hours were allowed to elapse before a diagnosis was made, not to mention the fact that during this time no treatment had been instituted. This time was used to obtain relatively useless X-ray plates, which when obtained added nothing to the indications for or against treatment: Second: In a case obviously suffering from a high intracranial pressure 10 cc. of cerebrospinal fluid was withdrawn by lumbar puncture previous to any cranial decompression. This subjected the patient to the danger of immediate death from herniation of the medulla in the foramen magnum, and the fact that this complication did not arise is due to pure chance. Third: The operative procedure was badly planned. The operation of choice would without question have been a suboccipital decompression, and this was ruled out only because of the patient's poor general condition. As a substitute a subtemporal decompression was performed. This I believe now to have been bad judgment. While a much less formidable procedure than a suboccipital operation, nevertheless, in view of the fact that the child was verging on, if not actually suffering from, an oedema of the medulla, and consequently even from the most favorable view point only in a condition to stand the minimum of surgical interference, even a subtemporal decompression should have been recognized as being of too formidable a nature. Either the case should have been classified at once as a case of medullary oedema, and allowed to die without operative interference of any kind, or granting, as was thought at the time, that the patient was suffering only from an extreme degree of medullary compression, which had not as yet developed into an oedema of this portion of the brain, operative interference should have been limited to a simple ventricular puncture and drainage of the excess cerebrospinal fluid followed later on by either a subtemporal or suboccipital decompression, depending upon the amount of improvement following the ventricular puncture.

I wish to stress particularly, however, that this patient was allowed to go sixty hours without treatment, and without a diagnosis, and that in consequence she passed through at least two stages of intracranial hypertension, during either of which she would have been a favorable subject for such operative intereference as would have offered at the worst an even chance of recovery.

The final stage of increasing intracranial pressure is that in which there is an oedema of the medulla itself.[24, 25] In this stage the regulatory centres of the medulla have become fatigued and the vasomotor stimulation will no longer raise the general arterial pressure sufficiently to force enough blood to the medulla to overcome the anæmia there. As a result the blood pressure drops rapidly while at the same time the pulse and temperature rise, the pulse becoming very weak and irregular, and the temperature reaching 105 and 106—the so-called brain temperature. The respiration also rises to 40 or beyond—oedema of the lungs develops, and the patient dies. In this stage nothing that can be done, no matter what it is, will save the patient. Consequently these patients should not be operated nor touched in any way; it merely hastens the end.

It can be seen from the above that the signs and symptoms—pulse, blood pressure, respiratory rate and mental state commonly used as indications governing diagnosis, treatment, and prognosis—all depend for their variations upon the basic factor of an increase in intracranial tension or pressure. If the intracranial pressure is measured, these subsidiary manifestations are not needed, and should not be regarded in any other light than as confirmatory evidence.

REFLEXES.

With regard to reflexes and their presence and absence, it is my opinion that they serve only as a guide to the point of maximum injury to the brain, and give us no indication whatsoever in regard to the patient's condition, and most certainly none in regard to the method of treatment.

BLOOD IN CEREBROSPINAL FLUID.

I have frequently heard it said that blood in the cerebrospinal fluid obtained by lumbar puncture means everything from a fracture of the base of the skull to a middle meningeal hemorrhage. I have further heard this referred to as an absolute indication, and also as an absolute contraindication to operation. Blood in the cerebrospinal fluid as obtained by lumbar puncture, providing it is not due to trauma from the puncture itself, means one thing and only one thing—namely—hemorrhage into the subarachnoid space. It has no bearing whatsoever on the line of treatment to be adopted. As a matter of fact and experience, blood in cerebrospinal fluid obtained by lumbar puncture usually means a laceration of the brain providing the injury has been to the head.

OTHER SYMPTOMS.

Bleeding from the nose, mouth, and ears is often considered important from the point of view of treatment. This is not so. In the first place extra-cranial lacerations of these cavities must be ruled out. Having done that—and it is surprising how often this simple complication to a head injury will occur—bleeding from the mouth is practically always due to bleeding from the posterior part of the nose, the blood having dropped into the back of the mouth instead of running out through the nostrils. Bleeding from the nose means usually a fracture through the cribriform plate, and indicates nothing except that the prognosis should be extremely guarded on account of the liability to meningitis. This is due to the extension of infection through this fracture from the septic cavities of the nose and mouth. Scrupulous care should be taken to prevent this if possible. Bleeding from the ear may mean a fracture of the petrous portion of the temporal bone, and may carry with it injury to the seventh and eighth nerves on this side, possibilities that should be carefully looked into at the time the patient is first seen. Otherwise this symptom indicates nothing in the line of treatment, and directly affects the prognosis only in so far as to offer the possibility of a basal meningitis arising from neglect to keep the ear clean.

SURGICAL SHOCK.

Any patient who has received an injury to the head severe enough to produce symptoms is in a state of surgical shock immediately following the injury. The condition and appearance of these patients in no way varies from that seen in a patient suffering from surgical shock following other severe injuries. Until these patients recover from this condition of shock, it is of no importance what other complications they may be suffering from. They must have immediate and careful treatment for their shock. This includes absolute quiet and rest together with warmth and fluids. If they are to have absolute quiet and are to be kept warm that means than an extensive neurological examination and a lumbar puncture cannot be done. Neither of these examinations should be carried out at this stage. If the patient will not recover from the surgical shock it is of no importance to treat his lacerated brain. On the other hand, if and when he does recover from his shock, then there will be plenty of time to treat his lacerated brain without exposing him to unjustifiable risks. Patients who have been subjected to severe head injuries usually recover from their shock in from two to eight hours depending on the extent of the injury, or else they have been so severely injured that they do not recover at all.

Careful observation of this rule in treating trauma to the head will save many patients who are now killed by too precipitate operating while they are still in a badly shocked condition.

An increase in intracranial pressure, then, is of paramount importance as an indication for operation in injuries involving the brain. From the foregoing we learn that there is a normal intracranial pressure of from 6 to 10 mm. Hg., depending upon the existence of a normal relationship between the secretion and absorption of the cerebrospinal fluid. The cerebrospinal fluid is secreted by the choroid plexus, and distributed for absorption throughout the spinal and cerebral subarachnoid space. From this space it is chiefly—in fact almost wholly—absorbed into the venous circulation. A rise in intracranial pressure occurs when either the rate of secretion is increased, or the rate of absorption interfered with. A rise in the pressure of the cerebral venous circulation interferes with the rate of absorption of the cerebrospinal fluid. This rise may be produced in a variety of ways varying from a more or less temporary congestion of the meninges, to large extra or intra-dural blood clots, and subarachnoid, subpial, or intra-cortical hemorrhages. Depending upon the location and extent of these hemorrahges, localizing symptoms, such as reflex changes, paralyses, etc., may be produced. Depending on the amount of increase of the cerebrospinal pressure systemic symptoms involving the pulse and respiratory rates, blood pressure, changes in the eye grounds, and mental state may be produced. All of these objective symptoms are inconstant and variable, and the determination of their degree cannot be carried out with any accuracy. The degree of rise of intracranial pressure, on the other hand, can be measured with absolute accuracy, and the amount of the increase above the normal gives a constant and invariable indication as to what operative procedure must be undertaken to correct the intracranial pathology. Intracranial pressure as measured by the Landon Spinal Manometer, reading above 10 and below 16 mm. Hg. calls for repeated lumbar puncture with drainage of sufficient cerebrospinal fluid to reduce the pressure to normal. Readings above 16 mm. Hg. call for decompression, preferably subtemporal. No individual suffering from an injury to the brain should be either treated or examined while in a state of surgical shock, and no such patient who can be demonstrated to have reached the final stage of intracranial hypertension—namely oedema of the medulla—should be operated upon, as the mortality in this latter class of case is 100% regardless of the treatment instituted. It is dangerous and often fatal to postpone treatment on a patient suffering from intracranial hypertension until X-rays of the skull are available. They can give no pertinent information that is not already at hand, and have no bearing on the operative indications of the case.

CONCLUSIONS.

1. The indications for operation in injuries involving the brain are three: compound fracture of the skull, depressed fracture of the skull, and a rise in the intracranial cerebrospinal fluid pressure.
2. The intracranial cerebrospinal fluid pressure depends on the relation between the secretory powers of the choroid plexus and the absorptive powers of the cerebral venous circulation.
3. Intracranial hypertension may and often does cause death in the absence of any injury to the bony coverings of the skull.
4. All cases that have received or that are suspected of having received an injury to the brain, no matter how slight, should, as soon as they have recovered from their surgical shock, have the pressure of their cerebrospinal fluid measured, and the treatment should be based primarily upon this finding alone.

REFERENCES.

1. William Sharpe: The Diagnosis and Treatment of Brain Injuries; 3, 1920.
2. Charles H. Frazier: Surgery of the Spine and Spinal Cord: 141 et sec., 1918.
3. Walter E. Dandy: Experimental Hydrocephalus, Annals of Surgery: LXX. 129, August, 1919.
4. Charles H. Frazier: Surgery of the Spine and Spinal Cord: 146, 1918.
5. Ibid., 144 et seq.
6. Ibid., 147.
7. Ibid., 150.
8. Ibid., 150.
9. Ibid., 150.
10. Ibid., 151.
11. William Sharpe: The Diagnosis and Treatment of Brain Injuries: 65, 1920.
12. Ibid., 54 et seq.
13. Keen's Surgery: 111, 190 et seq.
14. Charles H. Frazier: Surgery of the Spine and Spinal Cord: 148, 1918.
15. William Sharpe: The Diagnosis and Treatment of Brain Injuries: 50, 1920.
16. Keen's Surgery: 111, 195.
17. William Sharpe: The Diagnosis and Treatment of Brain Injuries: 50, 1920.
18. Keen's Surgery, 111, 195.
19. Ibid., 198.
20. Charles H. Frazier: Surgery of the Spine and Spinal Cord: 151, 1918.
21. Ibid., 773.
22. William Sharpe: The Diagnosis and Treatment of Brain Injuries: 51, 1920.
23. Keen's Surgery: 111, 196.
24. William Sharpe: The Diagnosis and Treatment of Brain Injuries: 52, 1920.
25. Keen's Surgery: 111, 197.

CONCERNING THE TYPE OF INJURY TO RENAL EPITHELIAL CELLS WHICH INCREASES THE SUSCEPTIBILITY OF THE CELLS TO THE ACTION OF THE GENERAL ANESTHETICS.

By Wm. DeB. MacNider, M.D., Chapel Hill. N. C.,

The Laboratory of Pharmacology of the University of North Carolina.

SEVERAL years ago certain studies[1, 2] were made concerning the relative toxicity of uranium nitrate in animals of different age periods. The observation was made that uranium nitrate, when given in a constant quantity per kilogram, was more toxic for old animals than for young animals and puppies. There occurred in the old animals a more marked disturbance in the animals' metabolism and, furthermore, the kidney injury in the old animals was of a severer type than could be demonstrated in the kidneys of young animals and puppies. The characteristic difference in the pathology of the kidney in these animals of different ages was the amount of stainable lipoid material which appeared in the renal epithelium. The puppies and young dogs showed stainable lipoid material in the form of dust-like particles and small droplets in the epithelial cells of the loops of Henle, while the older animals showed large droplets and fused masses of such material in this location and also much stainable lipoid in the highly specialized secretory cells of the convoluted tubules.

In later publications,[3, 4, 5] there has been shown to occur a marked difference in the ability of the animals of the two age groups to form urine and to respond to various diuretic solutions when the animals of the different age periods had been poisoned by uranium nitrate and anesthetized by ether, chloroform or by Gréhant's anesthetic. The young animals and puppies, during a period of anesthesia which lasted from one hour to two hours and forty-five minutes, formed urine and, furthermore, the kidneys of these animals remained responsive to the stimulation of various diuretic solutions as caffein, theobromine and theocin. The group of old animals, on the contrary, during a similar period of anesthesia, either formed less urine per minute or urine formation ceased. In such animals the urine-forming value of the above-mentioned diuretic solutions was negative. That part of the kidney mechanism on which these substances act failed to respond to the stimulation. This lack of response on the part of the kidney is not due to any failure in the systemic blood pressure of the anuric and non-responsive animals, nor is it due to any lack of response on the part of the peripheral vascular mechanism of the kidney to these solutions. The pathological changes in the kidneys of the anuric animals, which differentiates them from the kidneys of the young animals that form urine and respond to diuretic solutions, is found in the renal epithelium. In the former group of animals, in which the use of uranium had led to a marked accumulation of stainable lipoid material in the epithelium, the anesthetic has induced changes leading to the death of these cells. In the puppies and young animals in which, following the use of uranium, there was less accumulation of stainable lipoid in the epithelium, the changes of degeneration following the anesthetic are either absent or slight in extent. Death of the cells does not occur.

In more recent investigations[6, 7, 8] studies have been made of the amount and distribution of stainable lipoid material in the renal epithelium of naturally nephropathic dogs, and of the susceptibility of the kidneys of these animals to the general anesthetics. These studies have shown that the primary injury to the kidney in such animals takes place in the glomeruli, the tuft of capillaries located at the site of origin of each uriniferous tubule. The injury to these structures results in the formation of connective tissue between the capillary loops, the walls of which become thickened and finally obliterated. The earliest evidence of injury which can be detected in the renal epithelial cells lining these tubules is an accumulation of stainable lipoid material in the cells of the loops of Henle and, especially, in the secretory cells of the convoluted tubules. When kidney tissue from such animals, stained for lipoid material, is compared with kidney tissue from normal animals that serve as controls, the difference in the amount and location of the stainable lipoid material is striking. In the kidneys of normal animals such material is only found as dust-like particles and droplets in the loops of Henle. It has not been demonstrated in the convoluted tubule epithelium. In the naturally nephropathic animals stainable lipoid material is found in the loops of Henle as large masses, which may be of such size as to obscure the structure of these cells, and such material is, furthermore, found as granular particles and droplets in the convoluted tubule epithelium. Following the establishment of this difference in the amount and location of stainable lipoid material in the renal epithelium of naturally nephropathic animals, as contrasted with the stainable lipoid content of renal epithelial cells in normal animals, both types of animals were anesthetized by ether or chloroform in order to ascertain if any relationship existed between the amount of lipoid material in the renal epithelium of the kidney and the susceptibility of these cells to the action of the anesthetics. The normal control animals of these experiments have continued to form urine during the period of anesthesia, and when the kidneys were subjected to the action of such diuretic solutions as caffein, theobromine, 5. per cent. glucose and 0.9 per cent. sodium chloride solutions, there developed an increase in urine formation.

The naturally nephropathic animals that were anesthetized by the same anesthetics for the same length of time have shown a marked decrease in urine formation as compared with the control animals or they have ceased to form urine. When such animals are given the above-mentioned diuretic solutions, the solutions are found to have less effect than they have in normal animals. In those naturally nephropathic animals that were rendered anuric by the anesthetics these solutions are ineffective. No urine formation occurs. A histological study of kidney tissue obtained from the two groups of animals shows that the renal epithelium in the normal control animals has been unaffected by the anesthetics. The cells show no distinct evidence of degeneration. The renal epithelium in the naturally nephropathic animals, which was shown to contain a great increase in the amount of stainable lipoid in comparison with the normal epithelium, has shown, following the period of anesthesia, marked degeneration characterized by edema, vacuolation and death.

From the foregoing analysis of the experimental data presented in this paper, the following summary appears allowable.

The earliest evidence of injury to renal epithelium which is induced by uranium nitrate consists in an increase in the amount of stainable lipoid material in these cells. The amount of such material that accumulates in the epithelium is influenced by the age of the animal. In old animals there is a greater accumulation of such material in renal epithelial cells than occurs in young animals and puppies. When animals from these age groups are anesthetized by chloroform, ether or by Gréhant's anesthetic, the severity of the action of the anesthetic on the renal epithelium shows a parallel with the amount of stainable lipoid material that can be demonstrated in such cells prior to the use of an anesthetic. The kidneys of old animals, in which the use of uranium has resulted in a marked accumulation of stainable lipoid, show very marked changes of degeneration from the use of the anesthetics. The kidneys of the young animals and puppies, in which less stainable lipoid has accumulated in the epithelium, show little or no injury from the anesthetics.

A study of the kidneys of naturally nephropathic dogs indicates that the primary injury to the kidney develops in the glomeruli. The earliest evidence of the secondary injury to the renal epithelium consists in a marked accumulation of stainable lipoid material in these cells and, especially, the secretory cells of the convoluted tubules. When such naturally nephropathic animals are anesthetized by chloroform or ether, and the effect of these anesthetics on the kidney is contrasted with the effect of the same anesthetics on the kidneys of normal control animals, a parallel is found between the toxic action of the anesthetics for the two groups of animals and the amount of stainable lipoid that can be demonstrated in the renal epithelium. In the kidneys of the naturally nephropathic animals, in which the amount of stainable lipoid has greatly increased in the renal epithelium secondary to the glomerular injury, the anesthetics induce an early degeneration and death of these cells, and urine formation is arrested. In the normal control animals, in which only a small amount of stainable lipoid can be demonstrated in the loops of Henle, such degenerative changes do not occur, and these animals form urine during the period of anesthesia.

Some twenty years ago Hans Meyer[9, 10] and Overton[11], working independently, developed a theory, the so-called Meyer-Overton Law, which is used to explain the entrance of certain narcotic and anesthetic substances into the cells of the central nervous system. These investigators were able to show that the affinity which the methane group of anesthetic substances possessed for the central nervous system was dependent upon the partition coefficient of these substances between the watery blood plasma and the lipoids of these cells. As the solubility of the anesthetic substance increased for the lipoid material of the cell it became of greater anesthetic value.

From the observations which have been made in the present study concerning the stainable lipoid content of renal epithelial cells, the Meyer-Overton Law would appear to apply to cells in this location as well as in the central nervous system. The investigation indicates that, following the accumulation of lipoid material in the renal epithelium, these cells, on account of their increased lipoid content, take up more of the anesthetic substance than do normal cells and, as a result of more of the anesthetic substance entering the cells, degenerative changes develop which impair or suspend the function of these cells. A definite relationship exists between the amount of stainable lipoid material in renal epithelial cells and the susceptibility of the kidney to the toxic effect of the methane group of anesthetic substances.

REFERENCES.

1. MacNider, Wm. deB.: On the Difference in the Response of Animals of Different Ages to a Constant Quantity of Uranium Nitrate. Proc. Soc. Exp. Biol. and Med., Vol. XI, 159, 1914.
2. A Consideration of the Relative Toxicity of Uranium Nitrate for Animals of Different Ages. Jour. Exp. Med., Vol. XXVI, I, 1917.
3. MacNider, Wm. deB.: A Study of the Action of Various Diuretics in Uranium Nephritis. Jour. Pharm. and Exp. Therap., Vol. III, 423, 1912.
4. MacNider, Wm. deB.: A study of the Action of Various Diuretics in Uranium Nephritis, with Special Reference to the Part Played by the Anesthetic in Determining the Efficiency of the Diuretic. Jour. Pharm. and Exp. Therap., Vol. IV, 491, 1913.
5. MacNider, Wm. deB.: The Effect of Different Anesthetics on the Pathology of the Kidney in Uranium Nephritis. Jour. Med. Research, Vol. XXVIII, 403, 1913.
6. MacNider, Wm. deB.: A Pathological Study of the Naturally Acquired Chronic Nephropathy of the Dog. Part I. Jour. Med. Research, Vol. XXIX, 177, 1916.
7. MacNider, Wm. deB.: A Pathological and Physiological Study of the Naturally Nephropathic Kidney of the Dog, Rendered Acutely Nephropathic by Uranium or by an Anesthetic. Part II. Jour. Med. Research, Vol. XXIX, 199, 1916.
8. MacNider, Wm. deB.: A Preliminary Paper on the Relation Between the Amount of Stainable Lipoid Material in the Renal Epithelium, and the Susceptibility of the Kidney to the Toxic Effect of the General Anesthetics. Jour. Pharm. and Exp. Therap., Vol. XVII, 289, 1921.
9. Meyer, Hans: Zur Theorie der Alkoholnarkose. Arch. f. Exp. Path. u. Pharm., Vol. XLII, 109, 1899.
10. Meyer, Hans: Zur Theorie der Alkoholnarkose. Arch. f. Exp. Path. u. Pharm., Vol. XLVI, 338, 1901.
11. Overton: Studien über de Narkose, Jena, 1901.

ACUTE PUERPERAL INVERSION WITH THE REPORT OF A CASE.[*]

By Joseph P. Cohen, M.D., Boston.

In perusing the statistics of the incidence of acute puerperal inversion in the literature, the casual reader may be led to believe that it need only be given academic thought. But the one bit of enlightenment these figures should afford us is their lack of uniformity. In addition to this there appears to be a strong inclination on the part of authorities to believe that there are a number of cases which probably have occurred and still do occur that are not reported because of the culpable manœuvres inciting inversion. This is particularly true of the private practice of obstetrics done by untrained men and unskilled midwives among whom, should the patient succumb, death is probably attributed to another superimposed condition and the true situation not brought to light. This is, of course, a misfortune, the correction of which can only be carried out by educational methods and the recognition that acute puerperal inversion may result in the best hands and despite good care. Prompt diagnosis is of first importance; once this is accomplished, proper treatment should be instituted.

---

[*] Read by invitation before the Boston Obstetrical Society on October 25, 1921.

The following table shows the significance of the irregular incidence of acute puerperal inversion:

TABLE

Jardine	1 in	17,000 cases
Winckel	0 in	20,000 cases
Braun	1 in	250,000 cases
Denham	1 in	100,000 cases
Beckman	0 in	250,000 cases
Madden	1 in	190,000 cases
Jones	1 in	127,000 cases
Kehrer	1 in	2,000 cases
Ahlfeld	1 in	100,000 cases
Zangemeister	1 in	400,000 cases
Welponer	1 in	35,000 cases
Ferri and Lorini	1 in	1,440 cases
Engelmann	1 in	5,000 cases
Chetenham District Assoc.	1 in	6,300 cases
Aveling	1 in	100,000 cases
Total,	13 in	1,603,740 cases
Average,	1 in	123,364 cases

Inversion of the puerperal uterus may take place immediately after labor or hours afterward. Macfarlane[1] declares the condition may arise as late as the fifth day postpartum. It may synchronize with the delivery of the placenta or it may come about after the third stage is completed.

Inversion may be partial or complete. It may be recognized very early as a mere cup-shaped depression in the fundus[2] or as an indentation which has approached only as far as the external os of the cervix; or it may not be discovered until the entire body of the uterus is turned inside out with the cervix shutting down on the lower segment of the uterus. The most extreme result is the inversion of the vagina along with the uterus: in these cases the entire uterus lies outside the vulva and drags the vagina with it.

ETIOLOGY.

The etiology of acute puerperal inversion is assignable ordinarily to two factors: pressure from above and pulling from below, though these do not account for the spontaneous types of inversion which are produced independently of surrounding conditions and form a definitely recognized entity. Improper employment of Credé's method of expressing the placenta appears to be one of the common causes. Either the manœuvre is carried out while the uterus is in a state of relaxation or else before the placenta has had time to separate. Engelmann[3] emphasizes that Credé of the uterus with one hand may be done improperly, and mentions the technique laid down by Credé himself, who states that it is wise "to await the contraction of the uterus, then seize it so that the fundus lies in the hollow of the hand and the five fingers on all sides of the corpus, after which gentle pressure is exerted." Engelmann goes still further, stating that, except in those cases where the uterus is very small, he deems it expedient to employ both hands in executing the Credé manœuvre and by doing so, distributes the pressure evenly throughout the uterus and minimizes the possibility of indentation from a thumb or one finger. He urges that compression should not be exerted at the same place each time the Credé is attempted.

It is my personal belief that the expression of the placenta during the third stage of labor is so important that it should never under any circumstances be entrusted to a nurse or inexperienced assistant. The third stage of labor should be cared for entirely by the physician. There has been a tendency to pass the responsibility of the delivery of the placenta to the nurse, who may not only be unfamiliar with the correct method to employ in expressing the placenta, but who, not infrequently, does not know how to "follow the fundus" or, if she does, is unable to appreciate what is taking place during relaxation or contraction. I have, therefore, made it a rule to insist upon the management of the third stage of labor by the skilled assistant and where this has not been feasible, I have taken the responsibility in my own hands.

Townsend-Whitling and Glover[4] report a case of complete inversion of the parturient uterus which took place thirteen hours after a normal delivery and three hours after the efforts of a nurse to expel a clot from the uterus. This brings to my mind another teaching which is not without pernicious character: I refer to the encouragement often given a nurse to knead a uterus which does not contract well, to force clots out of the uterus by compression of the fundus. It is far better to watch the uterus by following it with the palm of the hand than to manipulate it vigorously. More ice, ergot and pituitrin should be employed postpartum to control a relaxed uterus and less manipulation.

The erect posture during labor and a short cord are predisposing causes of inversion.

Dighton and Collins[5] report a case of inversion occurring after the normal delivery of a child with the cord wound about the neck, trunk and extremities. It seems that a case such as this would suggest that great care should be exerted in slipping the cord over the head when the cord is about the neck of the fœtus. In this connection there should be less hesitation in overcoming this cord difficulty by severing the cord between two clamps before delivery is completed.

Deliberate traction on the cord from below is generally acknowledged an obstetrical crime, and so the manœuvre is probably rare. Despite this, it remains as an important mechanical factor in some cases of inversion. Von Näcke's case[6] was the result of attempted removal of an adherent placenta. In Culbertson's case[7], inversion was discovered on the third day postpartum. The placenta had been removed manually an hour after delivery because of severe bleeding, and the uterus packed. The fundus had been felt definitely above the symphysis. Despite this fact, the inversion followed the removal of the packing on the third day.

Spells of coughing or sneezing, and pressure on the fundus by the straining abdominal muscles of the patient have been mentioned as causes of inversion.

Von Micholitsch[8] is of the opinion that puerperal inversion, excepting the spontaneous type, can only occur with a completely relaxed uterus. H. W. Yates[9] believes that a localized atony or thinness of the uterine walls is a factor requisite for the production of inversion. He adds that the exciting causes are fundal implantation of the placenta, the presence of submucous fibroids during pregnancy and the weight of a large placenta, particularly when situated at the fundus. Berkeley and Bonney[10] are convinced that unusual laxity of the wall of the uterus is essential in all cases of inversion, and Shears[11] is equally firm in the conviction that a well-contracted uterus cannot become inverted.

Engelmann[3], who reports having seen two cases of acute puerperal inversion, believes there is a certain significance to the occurrence of both cases in war time. He declares that the famine years increased the disposition of the uterus to atony and quotes a table of figures to show that there was a greater tendency to uterine hæmorrhage in the years 1918-19 than in 1913-14.

On the other hand, Beckman[12] has made a careful study of the literature, and has been impressed by the relatively high percentage of spontaneous inversions that have occurred in which the uterine wall, though well contracted, has suddenly vanished under the hand. He states that Küstner, Leopold and Pulvirenti have confirmed this observation.

In the consideration of the etiology of acute puerperal inversion, it is interesting to note the manner in which inversion may take place. There is the fundal method of inversion due to an indentation produced in the fundus of the uterus from mechanical pressure exerted at this point or from an inherent relaxation at the placental site when the placenta is so situated. Once this indentation occurs, it assumes the same relationship to the rest of the uterus as a foreign body or tumor which the uterus endeavors to extrude. Jones[13] of Chicago makes the reasonable supposition that practically all cases of inversion begin at the fundus, because it is this area of the uterus which is exposed to most of the forces contributing to the production of inversion. James L. Huntington[14] is of the opinion that fundal implantation of the placenta is the most likely condition to produce inversion, and believes the view is strengthened by the rarity of this placental situation.

There are two other theories used to explain inversion. Lateral inversion is a possibility that must be borne in mind, although this is probably the rare type of inversion; I could find no cases in the literature that definitely suggested this method of inversion. Still, it is conceivable that lateral inversion may take place with one side of the uterus starting to invert and the fundus following. The third method of inversion was recognized by Aveling[15], and had a great deal of ardent support from I. E. Taylor[16], who believed that a majority of acute spontaneous inversions of the uterus took place by the cervical route; it was Taylor's idea that, as a result of the paralysis of the cervix brought about after delivery, the cervix would not shut down. The body of the uterus, contracting against the relaxed cervix, rolled out the anterior and posterior lips and descended through this collar. Of recent years this method of inversion has been mentioned but little in the literature. It should be given serious thought, however, when we consider the cases reported as spontaneous inversions during the progress of which the fundus has been made out from above without evidence of indentation.

Von Micholitsch[8] believes that though the fundal type of inversion is most common, yet inversion may take its origin at any other parietal region of the uterus. The spontaneous type of inversion is most apt to be cervical, according to this same writer. He is in substantial agreement with Taylor in the belief that there is an invagination of the contracted body of the uterus into the paralyzed cervix, difficult labors predisposing to this paralysis.

In 1884, J. C. Reeve[17] observed that in inversion of the uterus of the cervical type there is first a pouting of the cervix and then an eversion of the lips followed by a rolling out of the body of the uterus. This, too, is in agreement with I. E. Taylor. Crosse[18] as early as 1845 states that the evidence points to inversion commencing at the superior portion of the uterus—usually the fundus—still "it would appear very possible, in a mechanical view, to find the progress of the displacement to be from the cervix to the fundus."

SIGNS AND SYMPTOMS.

Shock and hæmorrhage—one or both—and occasionally sudden, sharp, severe pain constitute the triad of signs and symptoms that usher in inversion. A feeling of something descending in the vagina may be noted by the patient. But there are cases in the literature where all these significant symptoms have been absent. Knipe's[19] patient, who had a spontaneous inversion immediately after a normal delivery, suffered from neither shock nor hæmorrhage, and reposition was readily done without the administration of an anæsthetic or without great distress to the patient. According to Kerr[20], a case is related by Galabin in which there were no symptoms whatever either immediately after delivery or during the puerperium: the condition was only recognized after lactation had ceased and irregular hæmorrhages occurred.

Inversion must be kept constantly in mind in all cases of unexplained hæmorrhage, and should be verified by bimanual examination. In the fundal type of inversion, an indentation may be discovered in the uterus by palpation, or, if the inversion is complete, by a recognition of a deep depression in the pelvis which has taken the place of the fundus. In the cervical type, the characteristic convexity of the fundus will be noticed in the early phase of the inversion, and at this time there may be present only what appears to be a thickened cervix.

TREATMENT.

In the consideration of the treatment of inversion, immediate replacement by taxis from below is the general rule. The taxis should be begun at the cervical end of the inversion, and care must be taken not to employ too much force for fear of rupturing the uterus[21]. Pressure must be conducted to conform with the curve of the sacrum, otherwise the object of reduction will be defeated. The danger of rupture is diminished by exerting counter-pressure on the abdominal wall with one hand. Bad results in some cases have been due apparently to attempted reduction without the administration of an anæsthetic. This is important because of the added shock which may accompany reinversion. Dinnick[22] favors the reduction of acute inversion by the abdominal route; in his case, reported in the Proceedings of the Royal Society of London, intestinal obstruction was found complicating the inversion. Since there is a tendency to acute retention of urine, it is well to be certain the bladder is empty before attempting any procedure which aims at reduction.

It occasionally happens that the cervix shuts down on the neck of the inverted organ so as to defeat all efforts at reduction. Occurrence of gangrene may result under these circumstances and hysterectomy be required. Dilation of the cervix in cases of constriction, either by the abdominal route or from below, has not been very successful because a good deal of shock is thus produced. The employment of the Küstner operation, which entails an opening across the posterior cul-de-sac and a slitting of the posterior lip of the cervix, is best adapted to subacute or chronic inversion rather than the acute type. Reinversion is accomplished by this method after the posterior wall of the uterus is slit longitudinally. Spinelli's procedure is an adaptation of the Küstner operation; the anterior cul-de-sac is opened transversely, the bladder pushed out of the way and the anterior lip of the cervix split, after which the anterior wall of the uterus is divided in the median line within a centimeter or two of the fundus. After reinversion the uterine body is dragged through the cul-de-sac opening and the uterine incision closed with interrupted sutures; the uterus is then returned through the cul-de-sac incision, which is closed except at a point to allow for the introduction of a small wick for drainage. Like the Küstner operation, the Spinelli technique is best employed in chronic inversion.

Sometimes, immediate replacement may be unwise[23]. This is true where the condition of the patient is precarious enough to indicate that further manipulation may prove fatal. According to Engelmann[3], Zängemeister is of the opinion from his own experiences and accumulated data, that the shock should be the first thing to combat; he believes that immediate operation for replacement only too often will cast the chance for life on the wrong side of the balance. He has preferred to wait a period of hours before the possible shock of reposition is thrust upon the patient. The cases reported in which late treatment was carried out are impressive. G. E. Shoemaker[24] mentions a successfully treated case of acute inversion in which it was decided to take care of a frankly septic uterus by constant irrigation with boric acid before replacement was attempted. Barrie and Donaldson[25] have also told of a case where hæmorrhage during acute puerperal inversion was treated by the administration of ergot and pituitrin and the hot vaginal douche. Transfusion was then done, and the septic element which developed on the third day postpartum was treated by continuous irrigation with 1 to 4000 potassium permanganate solution. Reposition was done seven weeks later with the Aveling Repositor, and was accomplished in nineteen hours.

It is my own opinion that, in the long run, immediate replacement is the procedure of choice, since in a certain number of cases there is no shock from reposition and because it is the safest way to check hæmorrhage. In addition, according to modern notions, sepsis can be cared for after reduction and the danger of a possible gangrene of the uterine body, resulting from the constriction of the cervical ring, is removed.

PROGNOSIS.

The mortality rate of acute puerperal inversion is quoted as low as 14 per cent. and as high as 80 per cent. An average of 45 per cent. is probably fair. Hæmorrhage, shock and sepsis may all together cause the fatal result or only one of these factors may cause death.

CASE REPORT.

Mrs. S. R., a primigravida 21 years old, was delivered by low forceps six hours after labor began. Her condition was splendid all during labor, but the vigorous character of the second stage disturbed her so much that it was deemed fair to apply low forceps, especially since the head was in sight. Delivery was accomplished without untoward incident. A slight second-degree tear of the perineum was repaired with catgut. A half-hour later the placenta was delivered by Credé's method; I personally took care of the expression of the placenta. There was no unusual difficulty in accomplishing this, and the uterus relaxed and contracted in a normal way. In the removal of the placenta, it was observed that only half of the membranes were present, but as there was no undue bleeding it was decided to leave them alone. The patient was put back to bed with a fair pulse, rate 120.

Five hours after delivery I was called to the hospital because the pulse was rising and bleeding was more than usual. There was no evidence at this time of shock. I decided that probably the membranes that were left in the uterus were accounting for the hæmorrhage and, after bimanual examination, determined to explore with the large dull curette, since the cervix was shut down and I felt that dilating the cervix for digital exploration might produce more shock. The fundus felt low and not very well contracted. Ergot and pituitrin had been administered. On inspection, what I at first took to be cervix, was probably a beginning rolling out of the body of the uterus which was taking place by the cervical method of inversion. Introduction of the curette was done with little force. The depth to which the curette reached, led to the suspicion of perforation and the instrument immediately withdrawn. It was decided to pack the vagina, leave the patient temporarily alone and treat her for shock which at this time had become marked. Hæmorrhage prior to packing was profuse. The pulse was thready. Blocks were placed at the foot of the bed and morphine administered. The advisability of transfusion was considered, though it was believed that shock rather than hæmorrhage was the predominating feature. Everything was prepared for transfusion, however, and a group four donor was secured as this was found compatible with the blood of the patient. It was about nine hours later, on bimanual examination, that uncertainty was set aside and a definite diagnosis of inversion was made. The added possibility of perforation made it seem desirable to reduce the inversion by the abdominal route. The patient was practically pulseless at the time, with the heart rate 150 at the apex. Upon opening the abdomen, a complete inversion was observed with the cervical end of the uterus shut tightly down upon its body. Allis forceps were applied to either side of the uterine wall, and the body was gradually reinverted by alternating traction. No particular difficulty was encountered. After reinversion, there was discovered the evidence of a small perforation about one centimeter from the left tube, the site through which the curette had passed. There was no undue bleeding, so the perforation was just closed by two deep catgut sutures and peritonealized. The uterus was hard. A small wick was introduced through the abdomen leading to the site of perforation and the abdominal wound then closed in layers up to the exit of the drain. While the fascia was being sutured the patient was transfused by the Vincent tube method, receiving about 750 c.c. of blood. This proved palliative for both shock and hæmorrhage. The patient stood the operation very well. She was put back to bed and further treatment for shock carried out. The quality of the pulse was now quite satisfactory. There was no complaint of pain at any time. Evidence of sepsis developed in twenty-four hours but remained localized. The lochia was foul and the uterus tender. There was no nausea or vomiting at any time and very little distention. The drain was removed on the fifth day.

On the ninth day after delivery, the patient had a chill which lasted twenty minutes; the temperature arose to 104.6 and the respirations to 30. The patient looked anxious, but did not complain of pain. Physical examination revealed nothing important. In four hours the temperature dropped to 99, the pulse to 106, and the respirations to 20. The possibility of a small pulmonary embolus was thought of as the cause of the disturbance. The remainder of the convalescence was uneventful. The mother went home in three weeks and a half, and was discharged well with her baby. Vaginal examination six weeks after delivery showed an involuted uterus which was normal in size, position and mobility; the os of the cervix was closed, and the vaults failed to disclose anything unusual.

SUMMARY.

1. A study of the literature indicates that acute puerperal inversion occurs about once in 100,000 deliveries.

2. The chief etiological factors are: Improper use of the Credé method of expressing the

placenta, pulling on the cord, uterine atony, manual extraction of the placenta a paralyzed cervix, intra-abdominal pressure from sneezing or coughing, tumors situated at the fundus and fundal implantation of the placenta. There is a difference of opinion as to their relative importance.

3. The methods of inversion are fundal, lateral and cervical, but the occurrence of spontaneous cases emphasizes the importance of the cervical route as a result of paralysis of the cervix.

4. Prophylaxis should be carried out by personal management of the third stage of labor by the obstetrician, and the early use of ice, ergot and pituitrin, with less manipulation of the uterus after the delivery of the placenta.

5. In reducing the inversion by taxis, care must be taken to avoid rupture. Other operative procedures are indicated where this fails.

6. If shock and hæmorrhage are very severe, these should be treated in preparation for subsequent reduction.

7. The average mortality is about 45 per cent.

REFERENCES.

1. Macfarlane, W. D.: Inversion of the Uterus, with Notes of Six Cases. Glasgow M. J., 1917, 87, 193-205.
2. Evans, W. G.: Total Inversion of the Parturient Uterus. Lancet, 1920, 1, 713.
3. Engelmann, F. von: Zur Frage der Haufigkeit, Entstehung und Behandlung der Puerperalen Uterusinversion. Zentralblatt f. Gynak., 1920, 44, 337.
4. Townsend-Whitling and Glover: A Case of Acute Total Inversion of the Parturient Uterus. Lancet, 1920, 1, 495.
5. Dighton and Collins: A Case of Complete Inversion of the Uterus in Parturition. Journ. Obstet. and Gyn. Brit. Emp., October, 1905, 8, 250.
6. Nacke, W. von: Inversio Uteri Puerperalis Inveterata und die Konservierende Inversionsoperation nach Kustner. Zentralblatt f. Gynak., 1918, 42, 190.
7. Culbertson, C.: Inversion of the Uterus. Surg., Gyn., and Obstet., 1920, 30, 315.
8. Micholitsch, Th. von: Zur Aetiology der Uterusinversion. Zentralblatt f. Gynak., 44, 758.
9. Yates, H. W.: Inversion of the Uterus. Am. J. Obstet., 1919, 80, 712.
10. Berkeley and Bonney: The Difficulties and Emergencies of Obstetric Practice. 1915 ed., 179.
11. Shears, G. P.: Obstetrics, Normal and Operative. 1920 ed., 455.
12. Beckman, W.: Zur Aetiologie der Inversio Uteri Postpartum. Zeitschr. f. Geburts. u. Gynak., 1895, 31, 371.
13. Griffin, J. M.: Report of a case of Acute Inversion of the Uterus. Am. J. Obstet., 76, 639. (Quoting Jones of Chicago.)
14. Huntington, J. L.: Acute Inversion of the Uterus. Bost. Med. and Surg. Jour., 184, 376-380.
15. Aveling, J. H.: On Inversion of the Uterus. 1886, 9-25.
16. Taylor, I. E.: On the Mechanism of Spontaneous Active Uterine Inversion. 1872, 1-43. (Reprint from N. Y. Med. Journ., May, 1872.)
17. Reeve, J. C.: Moot Points in regard to Inversion of the Uterus. Gyn. Trans., Vol. 9, 1884. (Reprint.)
18. Crosse, J. G.: Essay on Inversio Uteri. 1845, p. 22.
19. Knipe, N. L.: Complete Inversion of the Uterus and Vagina Following Labor. Am. J. Obstet., 1917, 75, 1019.
20. Kerr, J. M. M.: Operative Midwifery. 1916 ed., 679.
21. Boyd, G. M.: Some Remarks on Inversion of the Uterus. Am. J. Obstet., 1919, 80, 161-166.
22. Dinnick: Inversion of the Uterus Complicated by Intestinal Obstruction. Proc. Roy. Soc. Med. Lond., 1917-1918, 11, 45.
23. Iurcev, G. von: Beitrag zur Puerperalen Uterusinversion. Wien. Klin. Wochnschr., 1918, 31, 1009-1011.
24. Shoemaker, G. E.: Inversion of the Uterus. Am. J. Obstet., 1917, 76, 268.
25. Barrie and Donaldson: Acute Inversion of the Uterus; Treatment by Blood Transfusion and Late Replacement. Proc. Roy. Soc. Med. Lond., 1921, 14, 207.

The American Association of Anaesthetists and the Mid-Western Association of Anaesthetists will hold a joint meeting in St. Louis, May 23–24, at Hotel Jefferson, the first three days of the A.M.A.

Current Literature Department.

ABSTRACTORS.

Gerardo M. Balboni
Laurence D. Chapin
Austin W. Cheever
Isador Coriat
Ernest M. Daland
Richard S. Eustis
Robert M. Green
John B. Hawes, 2d
John S. Hodgson
Fred S. Hopkins
Charles H. Lawrence
Herman A. Osgood
Francis W. Palfrey
Edward H. Risley
William M. Shedden
George G. Smith
John B. Swift, Jr.
Wilder Tileston
Bryant D. Wetherell

Bastedo's Symptom in Appendicitis.

Jerlov, E. (*Acta Chirurg. Scandinavica*, Vol. LIV, fasc. ii). Bastedo's Symptom consists of a sensation of pain in the region of the appendix when the colon, previously emptied, is inflated. Jerlov has refined the technic by attaching a manometer to the rectal tube, and by attaching a distensible bulb which prevents the escape of gas around the tube. Air is introduced into the bowel until pain is felt in the region of the appendix, or until the pressure equals 120 mm. mercury.

Ulcerative colitis, typhoid fever and dysentery are contraindications to the use of this test. Jerlov employed it on 100 cases with no injurious results. He emphasizes the fact that it should be employed with great caution. The results in his 100 cases follow:

1. Chronic catarrhal appendicitis—24 cases; 13 positive, 11 negative. Bastedo positive (average) at 50 mm. Hg.
2. Chronic appendicitis with acute catarrhal exacerbation—19 cases; 17 positive, 2 negative. Bastedo positive (average) at 32 mm. Hg.
3. Ulcerative or gangrenous appendicitis—14 cases; 13 positive, 1 negative. Bastedo positive at 31 mm. Hg.
4. Appendicitis with peritonitis—3 cases; 3 positive. Bastedo positive at 33 mm. Hg.

In 38 other cases with various intra-abdominal pathology, including four cases from which the appendix had been removed, Bastedo's sign was negative.

Jerlov believes that the sign is positive when the wall of the appendix is indurated, even if the induration is not inflammatory. (A carcinoma of the appendix gave a positive test.) If the appendix is bound down by adhesions, however, it will not respond to the test. [G. G. S.]

So-called Abacterial Renal Pyuria.

Söderlund, G. (*Acta Chirurgica Scandinavica*, Vol. LIV, fasc. ii). The writer reviews the literature on the subject of abacterial renal pyuria, and quotes at some length from the work of Runebergs. The latter had collected 56 cases exhibiting this condition; of these, 18 were cases of renal tuberculosis. Of the remaining 31, Runebergs had done nephrectomy in 10. Eight of these ten showed congenital abnormalities; three showed a follicular pyelitis; in five the ureter was thickened and adherent. Microscopically these cases showed pyelonephritis, and in some sections micrococci were found. Söderlund reports three cases of abacterial renal pyuria, all in otherwise healthy men. In all, the condition was bilateral. The symptoms were of gradual onset and consisted of frequency of urination, strangury and terminal hematuria.

The urine was turbid. Cystoscopy showed edema and redness of the bladder wall, with considerable fibrinous exudate. Ureteral specimens were turbid; the sediments showed pus and a large amount of fibrin. No bacteria could be found in stained specimens or by culture. Repeated search showed no tubercle bacilli.

In Case 1, Söderlund removed one kidney. It showed slight chronic interstitial nephritis, scattered glomerulitis, follicular pyelitis and a thickened pelvis and much thickened ureter. It was not specific. After the nephrectomy in Case 1, and after several years in Cases 2 and 3, the condition cleared up completely. This fact, the writer believes, ruled out tuberculosis. He is convinced that there was no gonococcus infection.

He believes there are two types of abacterial renal pyuria, the one due to a micrococcus which is not discovered in the urine; the other, perhaps, due to the elimination through the kidney of some toxin.

[G. G. S.]

CARCINOMA OF THE PROSTATE.

BARRINGER, B. S. (*Surgery, Gynecology and Obstetrics*, February, 1922) treats this condition largely from the point of view of radium. He analyzes about one hundred forty-five cases. He states in conclusion that in but two per cent. of cases carcinoma of the prostate seen at the Memorial Hospital was the disease apparently confined to the prostate alone: that routine prostatic examination of all patients beyond the age of fifty, irrespective of symptoms, is the only rational method whereby we can make diagnosis of prostatic carcinoma early in the disease: that the results of radium treatment of carcinoma of the prostate are superior to operative removal both in causing regression of the disease and in coping with urinary retention.

[E. H. R.]

HISTOLOGY AND MORTALITY IN CASES OF TUMOR OF THE BLADDER.

SCHOLL, ALBERT J. (*Surgery, Gynecology and Obstetrics*, February, 1922) writes as follows:

In 168 epithelial tumors of the bladder removed at operation were three benign papillomata. The three patients are alive on an average of five years after operation. In these one hundred and sixty-eight cases were seventy-one malignant papillomata and ninety-four solid carcinomata. Twenty-six (36.6 per cent.) of patients in the first group have been dead on an average of eleven months after operation, in contrast to sixty-seven (71.2 per cent.) of the latter group who died on an average of seven and one-half months after operation. Forty-five (43.4 per cent.) of the patients with malignant papillomata are alive on an average of three years and three months since their operation, in contrast to twenty-seven (28.8 per cent.) of the patients with solid carcinoma who are alive on an average of two years and three months. Forty-one and nine-tenths per cent. of all patients operated on for malignant papilloma have lived more than three years after operation, only 10.6 per cent. of patients with solid carcinoma have lived more than three years after operation. The incidence of recurrence following operation on patients for solid carcinoma is much greater than that for malignant papilloma. In a solid tumor the local extension is generally more advanced; the removal requires a more extensive operation and the immediate mortality is greater.

Squamous-cell carcinomata of the bladder are extremely malignant and rapidly fatal: five of six patients with tumors of this type died after a very short period of symptoms.

HISTOLOGY AND MORTALITY IN CASES OF TUMOR OF THE BLADDER.

Adenocarcinomata are about as severely malignant as papillomata. Of five patients, one died two years after operation, one had a recurrence in two years, three are well, two, six months and one, two years after operation.

Simple angiomata of the bladder may grow so large as to cause obstruction. In three cases, one child of seven died of hemorrhage from a tumor growing from the bladder to the rectum; a man aged seventy-six years was first seen in an inoperable condition; and a girl aged nineteen years had an extremely large tumor in the dome of the bladder. This was removed, and five years later the patient was perfectly well.

Myomata of the bladder often grow very large. There was one patient in the series with myoma. Eight years after the removal of a large tumor the patient was alive and well.

Generally myxomata occur in young persons. Two infants in the series had myxomata; both died shortly after operation.

Sarcoma is probably the rarest and most malignant of vesical tumors. It occurs in middle-aged persons, metastasizes extensively, and has a tendency to recur rapidly. The one sarcoma in the series was seen at an inoperable stage.

[E. H. R.]

SYNOVIAL MEMBRANE TUMORS OF JOINTS.

HARTMAN, FRANK W. (*Surgery, Gynecology and Obstetrics*, February, 1922) writes a very interesting article on this little-discussed subject. His article is illustrated with microscopic sections and case histories. He does not go into the literature on the subject, nor does he classify the various lesions. The question arising in face of knee-joint tumors is that of saving the limb and the function of the joint. It has been found that tumors of low-grade malignancy can often be entirely removed without recurrence and a functioning joint secured. These cases, however, are comparatively few, and most of them require resection of the joint.

[E. H. R.]

SACRAL ANAESTHESIA IN UROLOGY.

SCHOLL, ALBERT J., JR. (*The Journal of Urology*, August, 1921). An anaesthesia that will not appreciably increase the work of the kidneys, such as sacral anaesthesia, is a desirable asset. In the urological examination of any large group of cases there is a certain percentage requiring a complete anaesthesia of bladder and urethra. Spinal anaesthesia, while it affords excellent relaxation, induces too extensive anaesthesia for minor operations. It inevitably keeps the patient in bed for a certain time, and carries with it a definite element of danger if not handled by an operator skilled in its use. Etherization must be very deep before relaxation of the bladder is obtained, and like spinal, it necessitates hospitalization for the patient. The restlessness and movements of the patient under a general anaesthetic greatly interfere with the accuracy and expediency of the examination. Sacral anaesthesia has been found valuable in the examination of contracted, irritable bladders, particularly those of tuberculous origin. The insertion of radium needles into malignant prostates, fulguration of tumors of the bladder, urethral and ureteral dilatation, litholapaxy and other operations have been readily carried out under this anaesthetic. The injection is made by passing a thin, spinal puncture needle through the sacral hiatus; then 30 cc. of specially prepared solution of a mixture of novocain, sodium chloride, and sodium bicarbonate is injected. Care is necessary that this be entirely an extradural injection.

[B. D. W.]

THE BOSTON
Medical and Surgical Journal
Established in 1828

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

For three years James S. Stone, M.D.
Horace D. Arnold, M.D.
Channing Frothingham, M.D.
For two years Homer Gage, M.D., *Chairman.*
Edward C. Streeter, M.D.
Edward W. Taylor, M.D.
For one year William H. Robey, Jr., M.D.
Roger I. Lee, M.D.
Robert B. Osgood, M.D.

Editorial Staff.
David L. Edsall, M.D.
Walter B. Cannon, M.D.
Reid Hunt, M.D.
Robert W. Lovett, M.D.
Edward H. Nichols, M.D.
Francis W. Peabody, M.D.
John P. Sutherland, M.D.
S. Burt Wolbach, M.D.
George R. Minot, M.D.

Walter P. Bowers, M.D., *Managing Editor.*
George G. Smith, M.D., *Assistant Editor.*

Subscription Terms: *$6.00 per year in advance, postage paid for the United States. $7.56 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the printer with galley proof of paper. Upon written request, authors will be furnished free one hundred eight-page reprints, without covers, or the equivalent in pages in articles of greater length.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.

THE PRESENT STATUS OF PATHOLOGY.

Men of the best type are no longer being attracted into pathology as a life work, but only as a stepping-stone to some other branch of medicine, such as surgery. There are at least two reasons which may be assigned for this state of affairs. One is that the salaries paid to teachers of pathology are not adequate as compared with those received by clinicians, even by those engaged on the full-time basis. The other is that the rewards of research in pathology do not seem so many and so promising as in other, newer lines of investigation.

The young medical man, after years spent in acquiring his education, needs and wants money and, if he is scientifically inclined, a fruitful field for investigation. If he is to succeed, he must advertise himself, and the publication of research work is the one way in which, at the present time, he can most successfully do so. When a man is discussed for a position in a medical school, almost the only question asked is, "What has he published?" The longer the list of his published papers is, the better are his chances of obtaining the position. Nothing else counts for very much, least of all, teaching ability.

Pathology seems to be regarded by many as a finished, dead subject, like anatomy, with little or nothing left to discover. For the past generation or more, an offshoot of it, bacteriology, has held the floor. More recently, serology came to the front. At the present moment, chemistry is being actively exploited as the one field of investigation which is going to clear up all that is medically unknown.

Each line of work is, in turn, drained of its easy, obvious possibilities as the nuggets of gold were picked up by the early prospectors. Pathology has now reached the stage of hydraulic mining. The problems remaining to be solved require a high degree of ability and training, in addition to time and patience.

There are three more or less interrelated problems connected with the present status of pathology that deserve brief consideration, namely, teaching, hospital laboratories and research.

It goes without saying that all men who teach pathology should first have at least two years of thorough training under competent supervision, in the pathological laboratory of a hospital in order that they may see and study a large amount of pathological material derived from post-mortem examinations and surgical operations. They require special drill and experience in their own line of work, just as much as the men who become internes in medicine, surgery and the specialties, before they are qualified to give instruction to others.

Most men do not care for teaching. It is an art by itself with which but few are gifted, but a certain degree of success in the art may be attained by most men with proper coaching and training. To teach well is a very useful and needful accomplishment for medical instructors, because the students pay to be taught what is known about the various subjects studied and how best to make use of the knowledge acquired. Hence knowledge of his subject and ability to teach are probably the most important requirements of a professor in a medical school. Research ability has its use there, but plays a minor part.

To obtain well-trained men with teaching ability costs money, and medical schools are not inclined to pay the price. They usually will put in a good head of a department at a fair salary, but limit his budget and force him to obtain almost anyone available to fill each instructorship. This is not the best way to build up a strong department which should be constantly developing the teachers of the future.

There has arisen, within the last few years, but especially since the war, a great demand for pathologists for hospitals, partly owing, it is to be hoped, to a better appreciation of their usefulness to the clinician, but partly on account of the desire of the hospitals to be ranked in Class A. As a result, almost anyone, at the present time, who will call himself a pathologist, can obtain a position.

The demand far exceeds the supply. The salaries paid are out of all proportion to the actual pathological training and knowledge which many of the men have acquired; for example, from personal observation, $2000 and maintenance, $4000 and $5000 a year for recent graduates with only one year's training in a pathological laboratory. With such opportunities before them, few men will accept teaching positions in a school at one to two thousand dollars a year, or less.

There are, however, certain disadvantages connected with these newly created positions for pathologists. The incumbents are expected to know and to be able to do everything in pathology, bacteriology, serology, and clinical pathology, including blood chemistry. It is as though a man, after one year's surgical interneship, were compelled to do with equal authority and facility, surgery, gynecology, eye, ear, and the other surgical specialties. Naturally, it can't be done except in a very superficial way that often may have dangerous results.

These new openings in pathology, however, are going to force the medical schools to pay better salaries to their instructors of this subject or get along with very inferior men, except in so far as they obtain the occasional young man who looks ahead and plans for the future. As an independent pathologist working without supervision, he will not get far. His reward will be a large salary while young, with little prospect of much advancement or improvement in the future and small opportunity for investigation. If he really wishes to arrive somewhere near the top in his chosen field, he must work for years under good men, in hospital laboratories and in schools, teaching and investigating, otherwise he will not develop and broaden out.

In the larger hospitals, where a staff of assistants is available and the different lines of work are under the charge of separate men, the position of pathologist becomes most desirable. He is in the ideal position, at the fountain head of pathological problems which stimulate him to investigation. His energies are not exhausted by executive work and the teaching of undergraduates. He has all he wants in that line in training his assistants and such graduate students as he may admit to the laboratory. The salaries paid to well-trained men in these positions run as high as $8000, with a yearly budget of $45,000. This is the latest authoritative quotation, but larger amounts have been demanded.

Nearly thirty years ago a young medical graduate, now a professor in one of our best medical schools, formulated the following as the way to write a medical paper: three weeks in the laboratory, three months in the library, one hundred and twenty to one hundred and fifty abstracts from the literature. Living up to that principle made him and has paid him, but his contributions to medical science have not been noted for originality.

Since then, investigation — research work — has become the greatest fad of the medical schools. It counts above everything else. Unquestionably it has its uses,—to advance knowledge, to test out the teachings of the past, to correct mistakes. Unfortunately it has grown to be a great advertising scheme. Almost everything else is sacrificed to it. The ideal seems to be a line of research where every experiment performed can be rushed into print. Few men are content to work and wait until they can publish digested results. Investigations, of which all that is new could easily be presented in twenty to thirty pages, have been expanded to hundreds. The limit has already been reached, it is to be hoped, in luxurious monographs with wide margins and single illustrations to the page.

Few men are born investigators: the manufactured kind is common enough; fewer still are original. Medical schools make little attempt to obtain, develop and aid in every way the true variety. Instead, they often seem to prefer to seek out and reward those possessing self-assurance, aggressiveness, and ability to advertise.

To sum up these criticisms, which apply to other departments as well as to that of pathology: there is a strong tendency to pay too much attention to research work, too little to training students and assistants in fundamentals, the most important work of a medical school. Instruction and training should come first; when they are thoroughly organized and in perfect running order, research work may be considered.

There is still opportunity for discoveries in the field of pathology. The bacteriology and pathology of most of the commoner infectious diseases have been pretty thoroughly worked out; the essentials are known. But there still remain plenty of problems for study, as, for example, the causes of various chronic toxic lesions, such as those occurring in the liver, kidneys, pancreas, and blood-vessels. Sampson, of Albany, has just shown in his brilliant work on "Ovarian Hematomas of Endometrial Type," what a born investigator, with both clinical and pathological training, can do in the field of special pathology. The recent work on rickets, by a group of men in different fields of medicine working together, illustrates the value of coöperation. There is too little of it in our medical schools. Indifference or jealousy is much more common. The tendency with holders of research funds is to hire young men who have had little or no special training in any line of work and set them to covering all sides of any subject under investigation, so that any results obtained will redound solely to the credit and glory of the department concerned and of the man at the head of it. They would get farther and accomplish more if they worked in hearty coöperation with their expert colleagues.

A PHYSICIAN IN THE CABINET.

With the appointment by President Harding of Dr. Hubert Work of Pueblo, Colorado, as Postmaster-General, there is a physician in the Cabinet for the first time since Dr. James McHenry served as Secretary of War in the Cabinets of Washington and Adams. So long ago as 1892, Dr. H. O. Marcy, now of Cambridge, suggested to the Council of the Massachusetts Medical Society that it would be for the advantage of the medical profession to create the position of cabinet officer of public health. The Council decided that a position in the Cabinet would not increase the efficiency of a public health officer and shortly the agitation was dropped.

Dr. Work's appointment indicates that a physician may develop executive ability, although the practice of medicine *per se* does not conduce to such attributes of character. Dr. Work, after practising medicine in Colorado, managing a sanatorium in Pueblo, made himself useful to the National Republican Committee, showing there that he was an executive of a high order of merit. He presided over the meetings of the House of Delegates, the governing body of the American Medical Association, showing that he possessed the ability of a presiding officer, a rare quality in medical men; now he is president of that organization, and therefore is in touch with the larger policies of medicine as they apply to the public. He has had a year's experience as Assistant Postmaster-General under Will H. Hays, whose resignation went into effect March 4. It is to be hoped that he may improve the quality of the postal service, which has been poor since the war. Being in the President's official family, he will be able to join with Brigadier-General Sawyer, the Executive's personal physician, in giving advice on legislation touching the interests of the medical profession. We welcome the entrance of a physician to the official family of the President, and wish him all success.

MID-WINTER CLINICS OF THE MAINE PUBLIC HEALTH ASSOCIATION AND THE MAINE MEDICAL ASSOCIATION.

These exercises were held at the Eastern Maine General Hospital and the Chamber of Commerce, Bangor, Maine, on February 20 and 21, 1922. There were medical, surgical, venereal, x-ray, and orthopedic clinics, and a comprehensive program, including exhibits and scientific papers.

Massachusetts was well represented by Dr. A. K. Stone, who spoke on The Influence of Organization in Reducing Death Rate. Dr. Eugene R. Kelley discussed the Future Relationship of the Nursing and Medical Professions and Child Welfare Work as a Preventive Health Measure. Dr. Channing Frothingham gave the results of his study on The Relation of the Cults to General Medicine, and Dr. F. B. Lund presented the subject of An Ancient Conflict between Poetry and Anatomy.

Maine physicians also took prominent part in the presentation of papers and in discussions.

NEWS ITEMS.

Worcester State Hospital.—Dr. Oliver H. Stansfield was the speaker at the staff luncheon at this hospital, February 17. His subject was the "Diagnosis and Treatment of Heart Disease." On the second of March, Dr. Philip Cook spoke on the subject of X-ray Interpretation.

The regular meeting of the Worcester District Medical Society was held on Wednesday, March 8th, in the University Club rooms, 377 Main Street. Program: "Epidermophytosis," Dr. Charles J. White, Boston; "The Laboratory Investigation of Epidermophytosis," Dr. Arthur W. Greenwood, Boston; "The Late Syphilitic and Therapeutic Problem," Dr. George A. Dix, Worcester. The discussion was opened by Dr. Clara P. Fitzgerald, Worcester.

The monthly meeting of the Lawrence Medical Club was held February 27, with Dr. Leith, at the Y. M. C. A. rooms. The chairman for the evening was Dr. Look. Subject: "Pneumonia," by Dr. Edward Libby, Boston.

Massachusetts General Hospital.—A Staff clinical meeting was held Monday, March 13, 1922. Program: "Points Concerning Bile Pigment Metabolism," Dr. Chester M. Jones; "Studies of the Causes of Death in Early Infancy," Dr. Eli C. Romberg; "Results from Quinidin Therapy," Dr. Louis E. Viko; "Metabolism in a Case of Acute Myelogenous Leukemia," Dr. William G. Lennox.

Gift to the Medical School of Western Reserve University. — Samuel Mather has given $2,500,000 to this institution. This sum is in addition to previous gifts totaling $800,000.

University of Cincinnati College of Medicine.—A campaign is under way to secure an endowment fund of $216,000 for this college, which will make a total of $2,000,000 for

1920-1921. When this is secured, the Rockefeller Foundation will add $700,000, and the Carnegie Foundation $200,000. Ohio has ambitions for the creation of medical schools which will gain recognition throughout the world.

THE NEW FIELD SECRETARY OF THE NATIONAL COMMITTEE FOR THE PREVENTION OF BLINDNESS.—Mr. Lewis H. Carris, of Washington, D.C., has accepted the invitation of the National Committee for the Prevention of Blindness to become its Field Secretary, and has already entered upon his work. Mr. Carris was, until January 15th, Administrative Head for the Federal Board for Vocational Education, in charge of the administration of the Federal Vocational Educational Act and the Industrial (civilian) Rehabilitation Act.

As Field Secretary, Mr. Carris will assist local and state organizations interested in the campaign for the prevention of blindness and the conservation of vision. He will also assist school authorities in the organization of special classes for school children of such limited vision that they cannot profitably use the books and other educational equipment for normally sighted children.

DURING the week ending March 4, 1922, the number of deaths reported was 322 against 217 last year, with a rate of 21.98. There were 51 deaths under one year of age against 30 last year.

The number of cases of principal reportable diseases were: Diphtheria, 76; scarlet fever, 40; measles, 154; whooping-cough, 9; typhoid fever, 5; tuberculosis, 56.

Included in the above were the following cases of non-residents: Diphtheria, 12; scarlet fever, 6; measles, 1; tuberculosis, 6.

Total deaths from these diseases were: Diphtheria, 4; scarlet fever, 1; tuberculosis, 22.

Included in the above were the following cases of non-residents: Diphtheria, 2; tuberculosis, 2.

Influenza cases, 207; deaths, 9.

Lobar pneumonia cases, 93; deaths, 41.

HARVARD MEDICAL ALUMNI ASSOCIATION.—The Harvard Medical Alumni Association is planning to hold its annual meeting on Thursday, May 18th, 1922. Owing to the disarrangements during and after the late war, the affairs of the Association have been considerably in abeyance. This organization is now planning to resume activities. A very attractive program is being prepared for this coming annual meeting, and a copy of this in full detail will be mailed later. The meeting will begin in the morning, and there will be clinics at the Massachusetts General Hospital, Peter Bent Brigham Hospital, and Boston City Hospital. Following lunch and business meeting, a visit to the Harvard Medical School will be made, where the Society will be entertained by various heads of departments. There will be a dinner at the Harvard Club after this, and accounts given of the present activities of the Medical School, and its hopes for the future.

STATE HOSPITALS FOR CARE OF SICK AND FEEBLE-MINDED CHILDREN.—Under the Department of Public Welfare, the hospital for the care of sick children of normal mind will be completed early this year, relieving the State Infirmary at Tewksbury of about 100 children. Also, under the large building program at Belchertown, about 200 feeble-minded children will be transferred from Tewksbury, and with the six new units added to the present hospital accommodations at Tewksbury, the State Infirmary will have much-needed relief.

* * *

The Massachusetts Medical Society.

MEMBERSHIP CHANGES FROM JANUARY 1, 1922, TO MARCH 1, 1922.

INCLUDING deaths, action by the Council, namely, retirements, resignations, deprivations for non-payment of dues, transfers from one district to another without a change of legal residence; also changes of residence and address and errors in the Directory of January 1, 1922, as reported previous to March 1.

Compiled by the Secretary of the Society.

ALPHABETICAL LIST.

Adamian, Hovsep Garo, from Lawrence to Fresno, California, 2424 Ventura Ave.

Adams, William Carlton, East Taunton, now 375 Middleboro Avenue.

Allen, Gardner Weld, Boston, retired by Council, Feb. 1, 1922, 374 Commonwealth Ave. Mark name with dagger.

Ash, Richard Maurice, Quincy, now 2 Abbey Road.

Balcom, Kenneth Ira, Worcester, now 134 Lincoln St

Barach, Alvan Leroy, from Boston to New York City, 33 East 68th St.

Barney, James Dellinger, Boston, now 87 Marlborough St.

Bartley, John Joseph, Lawrence, now 334 Haverhill St.

Battershall, Joseph Ward, died at Attleborough, February 23, 1922, aged 79.

Berry, William Christopher, died at Roxbury, February 8, 1922, aged 62.

Blake, Gerald, Boston, now 311 Beacon St.

Blake, Harrison Gray, died at Woburn, January 26, 1922, aged 58.

Boivin, Omer Emèdeé, Fall River, 187 No. Main St.

Bowen, James Francis, St. Louis, Mo., U.S.P.H.S. Dispensary.

Briggs, Maurice Taggart. Delete second "v" from Drivve.

Brindisi, Rocco, Boston, 147 Richmond St.

Brownrigg, Albert Edward, Nashua, N. H., Highland Spring Sanatorium.

Bullard, Carleton Wheeler, Newburyport, now 194 High St.

Bunker, Henry Alden, Jr., from Medfield to Brookline; office, Boston, 240 Newbury St.

Burt, Clarence Edward. Delete "Westport, Office."

Butler, Patrick Francis, Boston, now 35 Bay State Road.

Butterfield, George Kittredge, from Hathorne to Providence, R. I., 305 Blackstone Boulevard.

Callahan, Henry Alphonsus, from Jamaica Plain to Medical Corps, U. S. Army.

Capeles, Thomas Francis, Haverhill, now 191 Merrimack St.

Carey, Bernard William, from Fitchburg to Winthrop; office, Boston, State House, Room 546.

Carroll, Henry Gerald, from Peabody to Salem, 309 Essex St.

Cary, Foster Harrington, Denver, Colo., now 266 Metropolitan Building.

Chapman, William Harden, Hingham, now Summit St.

Cheever, Austin Walter, Boston, now 21 Bay State Road.

Conner, Homer Leigh, from Haverhill to Medical Corps, U. S. Army.

Corcoran, George Bartlett, West Springfield, now 84 Park St.

Côté, Corinne Rhea, from Worcester to Boston, 483 Beacon St.

Coues, William Pearce, from Norfolk to Suffolk (Council), Boston, 31 Massachusetts Ave.

Councilman, William Thomas, retired by Council Feb. 1, 1922, 78 Bay State Road, Boston.

Crandall, Walter Midkiff, Medical Corps, U. S. Army.

Cruickshank from Cruikshank, Frank Sheppard, from Dorchester to West Roxbury, 53 Willow St.

Cummings, John Joseph. Delete dagger—he is not retired.

Curtis, Francis George, Chestnut Hill, Newton; office, West Newton, City Hall, Health Dept.

Curtis, Robert Dudley, from Boston to Newton Center; office, Boston, 355 Marlborough St.

Cushman, Howard Lewis, Methuen; office, Lawrence, 457 Essex St.

Daly, Jeremiah James, Andover; office, No. Andover, 26 Second St.

Derby, Joseph Patrick, Springfield, 525 Main St.

Devere, Fred Hewitt, Auburn, R. I., 667 Park Ave.

Dewis, John William, Brookline; office, Boston, 270 Commonwealth Ave.

Durrie, Anna Belle, resignation accepted by Council, Feb. 1, 1922. Cross off name.

Elkind, Henry Byron, Worcester, now 28 Pleasant St.

Enebuske, Claes Julius, from East Boston to Boston, 365 Massachusetts Ave.

Farrar, Lonnie Oliver, from State Farm to State Colony, East Gardner.

Finnegan, Philip Joseph, Salem, now 82 Washington Sq.

Fitts, Henry Bird, resignation accepted by Council Feb. 1, 1922.

Fitz, Reginald, from Roxbury to Rochester, Minn., Mayo Clinic.

Flagg, Elisha, Boston, from 92 to 192 Marlborough St.

Flood, Everett, retired, from Palmer to Mt. Dora, Florida.

1905 1922 } Flynn, Henry Lawrence, Jamaica Plain (Boston), 204 South St. Restored by Council Feb. 1, 1922.

Forster, Robert William, died at Lawrence, Feb. 7, 1922, aged 46.

Fremont-Smith, Frank, resignation accepted by Council, Feb. 1, 1922.

French, Charles Ephraim, Lowell, now 9 Central St.

Friedman, Harry Falk, from Boston to Allston; office, Boston, 270 Commonwealth Ave.

Frost, Harold Maurice, from Norfolk to Suffolk (Council), Boston, 374 Marlborough St.

Gaffney, Mary Evangeline, from Greenwood Mountain, Me., to Wallum Lake, R. I.

Gannett, William Whitworth, joined the Society in 1877, not 1887. Change date.

Garbelnick, David Abraham, from Bradford to Haverhill, 25 Lawrence St.

Généreux, Edmond Alfred, from Webster to Millville.

Gillon, Charles Joseph Carroll. "Carroll" omitted in Directory of Jan. 1, 1922.

Golden, Lazarus, from Norfolk to Suffolk (Council), Boston, 99 Green St.

Goodwin, Harold Merle, resignation accepted by Council, Feb. 1, 1922.

Gookin, Edward Richard, from Dorchester to Medical Corps, U. S. Army.

Graham, Douglas, retired by action of Council, Feb. 1, 1922. Mark name with dagger.

Green, John Orne, retired; died at Boston, Jan. 5, 1922, aged 80.

Greenwood, Sewall Elliot, Templeton, retired; died at Templeton, Feb. 5, 1922, aged 68.

Haley, William Thomas, from Salem to Boston, 531 Beacon St.

Hamblet, Mary Lucia, Wallum Lake, R. I. "Lake" omitted in Directory of Jan. 1, 1922.

Hamilton, Albert John Adams, now 270 Commonwealth Ave., Boston.

Hamilton, Robert DeLancey, Newburyport, now 220 High St.

Hamilton, William John, from Newton Center to Westwood, Westwood Lodge.

Hartnett, Edward Henry Lewis. Delete first "t." His name is Harnett.

Healy, William, West Natick. Delete "P. O. Box 223."

Hoberman, Samuel, Malden, now 115 Salem St.

Hussey, Earle Edward, from Newport, R. I., to Fall River, 422 No. Main St.

Jackson, Roy Chase, from Worcester to Middletown, Conn., Connecticut State Hospital.

Janjigian, Robert Rupen, from Bangor, Me., to Wilkes-Barre, Pa., Wilkes-Barre City Hospital.

Jewett, Everett Porter, from West Springfield to Three Rivers (Palmer), 100 Maple St.

Kassels. Change to Kassees.

Kelleher, Jeremiah Edward, Haverhill, now 120 Emerson St.

Kernison, Frederick Marshman, from Newton, N. H., to Boston, 654 Tremont St.

Khoury, Kamel, resignation accepted by Council, Feb. 1. 1922.

King, Nicholas James, Roslindale, now 63 Ashland St.

Lacey, Walter Hamer, Keene, N. H., now 85 Washington St.

La Liberté, Elie Joseph, Fall River, now 131 So. Main St.

Lavelle, Gertrude Helen, from Worcester to Natick, 1 Lincoln St.

Leith, Richard Bliss, Lawrence, now 575 A Essex St.

Limauro, Louis Herbert, Lynn, now 158 So. Common St.

Litchfield, William Harvey, from Brookline to Marblehead, 73 Front St.

Long, Rufus Wilfred, from Manchester, N. H., to Lowell, 226 Merrimac St.

Marcley, Walter John, Minneapolis, Minn., now 5110 Wentworth Ave.

Marvin, Harold Myers, resignation accepted by Council, Feb. 1, 1922.

McAllister, Frederick Danforth, Methuen; office, Lawrence, 301 Essex St.

McCarty, James Joseph, retired (Council), from Lowell to Minneapolis, Minn.

McDermott, Joseph Edward, from Boston to Charlestown, 296 Bunker Hill St.

McLaughlin, Arthur Otis, Haverhill, now 100 Emerson St.

Meredith, Florence Lyndon, from Watertown to Philadelphia, Pa., 1530 Spruce St.

Merrill, Everett Albert, resignation accepted by Council, Feb. 1, 1922.

Merritt, Robert Elmer, resignation accepted by Council, Feb. 1, 1922.

Mikolaitis, Casimir John, Lawrence, now 90 A Lawrence St.

Morse, Frank Leander, resignation accepted by Council, Feb. 1, 1922.

Mudge, Otis Pope, Amesbury, now 152 Main St.

Munro, Walter Lee, Providence, R. I., now 62 No. Main St.

Myers, Edmund, Boston, now 661 Tremont St.

Nye, Robert Nason, from Boston to Forest Hills (Boston), State Antitoxin and Vaccine Laboratory.

Ober, Frank Roberts. Change year of entrance to Society from 1901 to 1914.

Oberg, Frank Thorwald, resignation accepted by Council, Feb. 1, 1922.

O'Connell, Andrew Edward, Worcester, now 6 High St.

O'Connor, Joseph William, Worcester, now 208 Highland St.

Oviatt, George Alexander, of South Sudbury, died at Waltham, Feb. 26, 1922, aged 72.

Parris, Roland Oliver, from Brookline to Waban (Newton); office, Boston, 636 Beacon St.

Pattrell, Arthur Ellis, was transferred from Worcester to Suffolk by Council, Feb. 1, 1922. No change of address.

Parsons, Azariah Worthington, Mexico City, now Ave. Madero, 10.

Pearl, Samuel Maurice, from Norfolk to Suffolk (Council), Boston, 37 Chambers St.

Penhallow, Dunlap Pearce, Medical Corps, U. S. Army.

Phillips, Karl Tristram, resignation accepted by Council, Feb. 1, 1922.

Pigeon, James Cogswell Du Maresque, retired by Council, Feb. 1, 1922. Mark name with dagger.

Piper, Frank, from Boston to Cambridge; office, Boston, 53 Mt. Vernon St.

Ratté, Arthur Andrew. Delete accent over "e."

Reid, William Duncan, Boston, now 21 Bay State Road.

Robinson, William Perry, change from Haverhill to Ayers Village (Haverhill)

Rockwell, Llewellyn Harrison, Boston, now 479 Beacon St.

Round, Arthur Morey, retired by Council, May 31, 1921. Mark with dagger.

Rowley, William, died at Lanesville, Jan. 29, 1922, aged 57.

Saunders, Sallie Harding, from East Boston to Arlington, Office, East Boston, 103 Cottage St.

Shaw, John William, Newburyport, now 7 Orange St.

Sheehan, Edward Bernard, from Brookline to Boston, 520 Beacon St. (Council).

Sherburne, Andrew Edward, from Dorchester to Portsmouth, N. H., Islington Rd.

Smith, Lawrence Weld, Brookline; office, Roxbury, 40 Wigglesworth St.

Smith, Richard Ilsley, Boston, Boston City Hospital.

Smith, Walter Anson, retired by Council, Feb. 1, 1922. Mark with dagger.

Stack, John Joseph; deprived of the privileges of fellowship by Council, Feb. 1, 1922.

Stone, Ralph Edgarton, Beverly, now 221 Cabot St.

Street, Lionel Alexander Burnet, resignation accepted by Council, Feb. 1, 1922.

Sullivan, William Joseph, Lawrence, 273 Haverhill St. Delete "N" from before Haverhill.

Supple, William Raymond, Cambridge; office, Boston, 362 Commonwealth Ave.

Titus, Raymond Stanton, from Boston to Jamaica Plain; office, Boston, 65 Bay State Road.

Tolman, Henry. Delete "Jr." from his name.

Tuttle, George Thomas, Milton, 110 Highland St., not Ave.

Vaughan, Jonas Hobart; resignation accepted by Council, Feb. 1, 1922.

Vivian, William James, from Worcester to Jamaica Plain (Boston), 220 South St.

Vose, Robert Henry, from Norfolk to Suffolk (Council), Boston, 443 Beacon St.

1910} 1922} Wallace, Harold Lowe, Allston (Boston), 56 Park Vale Ave., restored by Council, Feb. 1, 1922.

Weissman, Ruth, from Long Island to Roxbury, 45 Townsend St.

Welles, Edward Sawtelle, resignation accepted by the Council, Feb. 1, 1922.

Wheeler, Charles Douglas, Worcester, now 340 Main St.

Wheeler, Lucia Anna, from Waterville, Me., to Wernersville, Pa., State Asylum.

Whelan, Charles, Hingham; office, Boston, now 375 Commonwealth Ave.

Williams, Carl Alonzo, Worcester, now 3 Irving St.

Winestine, Frederica, resignation accepted by Council, Feb. 1, 1922.

Winsor, Allen Pellington, Brookline; office, Boston, 402 Marlborough St.

Wood, Leonard, from U. S. Army to Manila, Philippine Islands.

Wood, William Franklin, from Brighton to Palmer, Monson State Hospital.

Local List.

Allston. Add to list the names of Friedman, H. F., and Wallace, H. L.

Andover. Mark name of Fuller, J. R., with dagger.

Arlington. Add name of Saunders, Sallie H.

Attleborough. Delete name of Battershall, Joseph W.

Boston. Mark Allen, G. W., with a dagger; also Councilman, W. T. (Council). Delete the following names: Barach, A. L.; Fremont-Smith, Frank; Green, J. O.; Haggert, G. E.; Irving, F. C.; Nye, R. M.; Piper, Frank; Stack, J. J.; Weissman, Ruth; Welles, E. S.; Winsor, A. P. Add the following names: Coues, W. P.; Enebuske, C. J.; Frost, H. M.; Golden, Lazarus; Haley, W. T.; Kassees instead of Kassels; McPherson, G. E.; Pearl, S. M.; Sheehan, E. B.; Smith, R. I.; Vose, R. H.

Bradford. Delete Garbelnick, D. A.

Brookline. Delete these names: Chase, H. L.; Coues, W. P.; Frost, H. M.; Litchfield, W. H.; Sheehan, E. B. Add these names: Bunker, H. A., Jr.; Irving, F. C.; Winsor, A. P. Mark name of Douglas Graham with a dagger. (Council, Feb. 1, 1922.)

Cambridge. Add name of Brides, A. E., and change first initial of Campbell, G. M., to C.

Charlemont. Add name of Parker, W. A.

Charlestown. Add name of Hogan, D. J.

Dorchester. Delete names of Cruikshank, F. S., and Pearl, S. M., also first letter "t" from Hartnett. Add name of Kandib, A. H.

East Boston. Delete name of Enebuske, C. J.

East Gardner. Add this town heading and enter Farrar, L. O. (It has no Post Office.)

Fall River. Add name of Hussey, E. E.

Fitchburg. Delete name of Carey, B. W.

Forest Hills. Add name of Nye, R. N.

Hanson. Add heading (See South Hanson).

Hathorne. Add name of Chase, H. L.

Haverhill. Delete Conner, H. L., and accent over "e" in Ratté. Add name of Garbelnick, D. A.

Hopedale. Delete Saunders, Sallie H.

Jamaica Plain. Delete Berry, W. C., and Callahan, H. A. Add Flynn, H. L.

Lancaster. Add Bartol, E. F. W.
Lanesville. Delete Rowley, William.
Lawrence. Delete Adamian, H. G., and Forster, R. W.
Lowell. Delete McCarty, J. J. Add Long, R. W., and Markham, E. W.
Lynn. Delete Merrill, E. A.
Marblehead. Add Litchfield, W. H.
Medfield. Delete Bunker, H. A.; Kandib, A. H.; McPherson, G. E.
Millville (*Worcester*). Add this heading and enter Généreux, E. A.
Milton. Delete Vose, R. H.
New Bedford. Change initials of Burt from E. W. to C. E.
Newbury (*Essex North*). Add this heading.
Newburyport. Change initials of Morse, F. D., to F. O.
Newton Center. Delete Hammond, W. J.
Palmer. Delete Flood, Everett, and add Wood, W. F.
Peabody. Delete Carroll, H. G.
Roxbury. Put dagger before name of Pigeon, J. C. D. (Council). Delete Prenn, Joseph. Add Weissman, Ruth.
Salem. Delete Haley, W. T Add Carroll, H. G.
Somerville. Delete Morse, F. L.
Southbridge. Delete Fitts, H. B.
South Sudbury. Delete Oviatt, G. A.
Springfield. Delete Koplin, Harry.
State Farm. Delete Farrar, L. O.
Taunton. Delete Ripley, H. G.
Templeton. Delete Greenwood, S. E.
Three Rivers. Add Jewett, E. P.
Waban (*Newton*). Add Parris, R. O.
Watertown. Delete Meredith, F. L.
Webster. Delete Généreux, E. A.
Westport. Change initials of Burt, from C. E to E. W.
West Roxbury. Add Cruickshank, F. S.
West Springfield. Delete Jewett, E. P.
Westwood. Add Hammond, W. J.
Whitman. Add Boch, Joseph.
Winthrop. Add Carey, B. W.
Woburn. Delete Blake, H. G.
Worcester. Delete dagger from name of Cummings, J. J. Delete names of Jackson, R. C.; Phillips, K. T.; Winestine, Frederica. Add Prenn, Joseph.

NON-RESIDENTS, BY STATES AND COUNTRIES

California. DeLangle, C. P., now Yountville. Delete Street, L. A. B. Add Koplin, Harry, Los Angeles.
Connecticut. Add Jackson, Middletown.
Florida. Add Flood, Everett, retired, Mt. Dora. Delete Vaughan, Jonas.
Kansas. Delete Oberg, F. T.
Maine. Delete Gaffney, M. E.; Janjigian, R. R; Wheeler, L. A.
Maryland. Delete Simmons, R. H.
Minnesota. Add McCarty, J. J., Minneapolis.
New Hampshire. Add Sherburne, A. E., Portsmouth.
New York. Add Barach, A. L., New York City; Haggart, G. E., New York City.
Pennsylvania. Add Janjigian, R. R., Wilkes-Barre; Meredith, F. L., Philadelphia; Wheeler, L. A., Wernersville.
Rhode Island. Delete Hussey, E. E. Add Butterfield, G. K., Providence; Gaffney, M. E., Wallum Lake. Add "Lake" to "Wallum," after Hamblet, M. L.
Medical Corps, U. S. Army. Make title as shown. Delete Wood, Leonard. Add Callahan, H. A.; Conner, H. L.; Crandall, W. M.; Gookin, E. R; Penhallow, D. P.; Simmons, R. H.
Foreign. Delete Boch, Joseph. Add Wood, Leonard, Manila, P. I.
Address Unknown. Delete Markham, E. W.; Merritt, R. E.; Parker, W. A.; Penhallow, D. P.
Wanted—Addresses of Azadian, David George; Bardwell, Frederick Albert; Bolduc, Alfred George; Gwynne, Samuel Carlton; Lawlor, John Charles.

Changes of Address and Errors in the Directory Should be Sent to the Secretary, 42 Eliot Street, Jamaica Plain.

◆

Miscellany.

LEGISLATIVE MATTERS.

The Constitutionality of the Sheppard-Towner Act.

An order has been introduced into the State Legislature by Eben S. Draper, calling for the opinion of the Attorney-General as follows:

Whereas, There is now pending in the General Court a bill, House, No. 181, being "An Act accepting the provisions of an act of Congress relative to the promotion of the welfare and hygiene of maternity and infancy, and providing for coöperation with the federal government"; and

Whereas, Doubt exists as to the constitutional right of the federal Congress to enact said federal act, being the Sheppard-Towner Act, so-called; therefore be it further

Ordered, That the Attorney-General is hereby requested to transmit to the General Court his opinion on the following questions:—

(1) Is the act of Congress, approved Nov. 23, 1921, entitled "An Act for the promotion of the welfare and hygiene of maternity and infancy, and for other purposes," within the constitutional powers of the federal government?

(2) Would the Commonwealth of Massachusetts, by the acceptance of said act, waive its rights as a sovereign State to contest the constitutionality of said act before the courts of the United States?

(3) If, in his opinion, said act is unconstitutional, what procedure should the Commonwealth now adopt to raise the question of constitutionality before the federal courts and to protest against the collection of money in this Commonwealth by federal taxation to provide funds for the operation of said act?

Speaking to this order, Henry L. Shattuck, in the course of his address, said:

"In the fiscal year ending June 30, 1916, Congress appropriated for the expenses of the Federal Government $678,677,859. In the current fiscal year the appropriations total $3,960,364,621, or over six times the appropriations of 1916. The internal revenue taxes alone collected by the Federal Government in

Massachusetts in 1921 amounted to $259,865,213.85,—over a quarter of a billion dollars in one year, and a sum over six times larger than the total of $39,736,266.33 appropriated in 1921 for running our entire State Government.

"Formerly the main sources of Federal revenue were from the tariff and from liquor and tobacco. The burden of these taxes rested upon the several States substantially in proportion to population and consumption.

"Today the main sources of Federal revenue are the individual and corporate income taxes and the inheritance tax. The burden of these rests with peculiar weight upon Massachusetts and the other highly developed industrial States, and it appears that much of the money collected by the Federal Government in Massachusetts is expended upon work in other States. This constitutes a great drain upon the resources of Massachusetts, and adds to the cost of living of all her inhabitants, whether they directly pay much or little in taxes.

"Let me illustrate the statement that the burden of these taxes falls with special weight on Massachusetts. While, as I have said, Massachusetts turned over to the Federal Government, in 1921, the huge sum of $259,865,213.85, Alabama, with a population nearly two-thirds that of Massachusetts, contributed only $18,429,531.41, or about 7 per cent. of the amount collected in Massachusetts, and Mississippi, with a population nearly one-half that of Massachusetts, turned over only $8,996,571.95, or less than 4 per cent. of the amount collected in Massachusetts.

"Massachusetts should therefore consider with special care all projects for Federal activities, involving, as they do, the expenditure of large sums of money.

* * * *

"To reduce the problem to its simplest terms, let us suppose that the sole function of the United States was road building, and that $100,000,000 was annually collected by income taxes, of which $5,000,000 came from citizens of Massachusetts, but that only $2,000,000 was spent on Massachusetts roads. It is obvious that Massachusetts loses $3,000,000 on the transaction, and would be $3,000,000 better off if it built its own roads with its own money.

"We are told that if we will appropriate a certain sum the Federal Government will 'give' us an equal sum, but this Federal money is not really a gift. It is a return of, perhaps, 30 cents on each dollar of additional Federal taxes collected from our citizens to meet the total outlay among the several States. By every such transaction we lose, and the majority of the States gain.

"The second part of the order raises the question of the constitutionality of these practices, and, more specifically, of the Sheppard-Towner Act, a bill for the acceptance of which is now before us. This act gives outright to each State the specific sum of $10,000 in the first year, and $5,000 thereafter, irrespective of population, and supplements this with a further sum of $1,000,000, to be apportioned among those States which appropriate a like amount, $5,000 to each, and the balance according to population. The expense rests with special burden on Massachusetts and the other highly developed industrial States. For example, Nevada, with a population of only 77,000, gets the same specific appropriation as Massachusetts, with a population of nearly 4,000,000; and Mississippi, which pays less than 4 per cent. of the internal revenue paid by Massachusetts, but has a population nearly half that of Massachusetts, gets the same specific appropriation as Massachusetts, and in addition gets an appropriation based on population nearly one-half as large as the appropriation based on population apportioned to Massachusetts. The fact is that a very small group of States, of which Massachusetts is one, pay most of the bills for the entire enterprise. The great majority of States get more than they contribute, and no wonder they are enthusiastic about this method of legislation.

* * * *

"We are dealing with the fundamental question of the power of the Federal Government, a question which transcends in importance any specific piece of legislation which may be offered for our acceptance.

"By Article X of the amendments to the Constitution, it is provided that the powers not delegated to the United States by the Constitution, nor prohibited by it to the States, are reserved to the States, respectively, or to the people. In other words, the powers of Congress are strictly limited to those delegated by the Constitution.

"Unlike the Federal Highway Act, which may be supported by virtue of the specific delegation to Congress by the Constitution of the power to establish post-offices and post-roads, and to regulate commerce, no specific power can be found in the Constitution to support the Sheppard-Towner bill. If constitutional, it must be supported solely on the power to lay and collect taxes, etc., to pay debts and provide for the common defence 'and general welfare of the United States.' But this clause gives Congress no power to legislate on any and all subjects it deems to be for the general welfare. Should any such construction be adopted, the very existence of the States would be threatened. Local self-government would be at an end. Congress would wield the supreme power. Every activity of our lives would be regulated from Washington. And we should soon be in the condition of France, where every detail of government is regulated from Paris, and which, in treatises on government, is cited as a horrible example of bureaucracy gone mad. In fact, by reason of the immense size of our country, and the great diversity in climate, needs, and conditions, the evils of bureaucracy would be far greater.

"It is high time that we should put this Federal power to the test."

Since this was written, the Committees on Public Health and Public Welfare, sitting jointly, have voted against the acceptance of the Sheppard-Towner Act.

The Spencer bill, providing for maternity benefits, also failed to secure support of the committee. This probably means that both bills relative to maternity and infant welfare will not be enacted in this session of the legislature.

MEETING OF THE ASSOCIATION OF ASSISTANT PHYSICIANS OF MASSACHUSETTS DEPARTMENT OF MENTAL DISEASES.

The fifty-third meeting of the Massachusetts Association of Assistant Physicians of the Department of Mental Diseases was held at the Psychopathic Hospital, Boston, Mass., on March 1, 1922. Forty-nine members were in attendance. At the request of Dr. H. G. Ripley, wives of members—some twenty in number—were present. After inspection tour through the institution, a buffet luncheon was served at 1 o'clock P.M. At 2 P.M., the meeting was called to order by the President, Dr. R. M. Chambers, and after a short business meeting, the program was presented by the Staff of the Psychopathic Hospital. Doctors H. G. Solomon and L. J. Thompson were in charge of the clinic. The first case demonstrated the technique of intraventricular puncture with drainage and introduction of Swift-Ellis serum. In Case 2, cistern puncture was made and the same procedure followed. Case 3 showed intravenous injection of arsphenamine by syringe method, followed by spinal drainage, continued until negative pressure was produced. Two greatly improved cases in general paralysis, one luetic encephalitis and two cases of lethargic encephalitis, with Parkinsonian syndrome, were then presented. Meeting adjourned.

N. A. Dayton, *Sec. and Treas.*

DEPARTMENT OF PUBLIC HEALTH LABORATORY IN THE WESTERN PART OF THE STATE.

Chapter 4 of the Resolves of 1921 contained a resolve for the investigation as to the necessity of establishing and maintaining a laboratory in the vicinity of Springfield.

The purpose of this resolve was to ascertain the possibility of more prompt service in reporting on specimens sent for diagnostic purposes. This work has been attended to by the Department of Public Health and the results appear in three conclusions, as follows:

1. It appears that a great economic gain would be effected by the food and drug inspectional work.
2. There would be in all probability an increase in the distribution and use of biologic products.
3. No great saving in time would be effected by the use of a bacteriological laboratory in the western section of the State over the use of the present laboratory facilities of the Department.

Inasmuch as the establishment of a laboratory for bacteriological diagnoses of communicable disease was the primary object in seeking the establishment of a laboratory in the western section of the State, we now recommend that this be not done at this time in view of the facts herein stated.

DEATHS FROM LETHARGIC ENCEPHALITIS ("SLEEPING SICKNESS").

The Department of Commerce, through the Bureau of the Census, has today (March 8, 1922) issued a statement showing the number of deaths in 1920 from lethargic encephalitis, which is often called, in this country, "sleeping sickness," although the true "sleeping sickness" is a very different disease, found, principally, in Africa.

In 1920, in the death registration area of the United States, lethargic encephalitis was given as a cause of death on 1,505 death certificates, as against 589 in 1919, giving mortality rates, respectively, of 1.7 and 0.7 per 100,000 population. Deaths from this cause were reported for every state in the registration area, except Delaware; the largest number in any one state was 364, reported for New York State, and giving a rate of 3.5 per 100,000 population.

Cities are credited with 1.129 of the 1,505 deaths, and rural sections with 376, with rates, respectively, of 2.6 and 0.8 per 100,000.

The white population, with 1,453 deaths from lethargic encephalitis, has a rate of 1.8 per 100,000 population, while the colored population, with only 52 deaths, has a rate of 0.7. Males and females contribute about equally to the total deaths from this cause, with 781 males and 724 females.

More of these deaths appear for persons aged 20 to 29 than for any other age group, though nearly as many are found for the age groups 30 to 39, and 40 to 49; and no age group escapes entirely.

NATIONAL LEGISLATION.

COMPENSATION OF PHYSICIANS AND NURSES.

THE Joint Congressional Committee which has been at work for several weeks drafting a bill to readjust the pay of officers in the Army, Navy, and Public Health Service, has agreed upon the terms of this legislation.

The following table summarizes the provision for base service pay of officers. In it we use the title of officers in the Public Health Service, with the corresponding army grade in parentheses:

Assistant Surgeon General (Colonel)	
Over 26 years' service	$4,000
First appointment above captain	4,000
Appointed under Sec. 24, Act June 4, 1920	4,000
Less than 26 years' service	3,500
Senior Surgeon (Lieutenant Colonel)	
Over 30 years' service	$4,000
Over 20 and less than 30 years	3,500
First appointment above second lieutenant	3,500
Appointed under Sec. 24, Act June 4, 1920	3,500
Less than 20 years' service	3,000
Surgeon (Major)	
Over 23 years' service	$3,500
Over 14 and less than 23 years' service	3,000
First appointment above second lieutenant	3,000
Appointed under Sec. 24, Act June 4, 1920	3,000
Less than 14 years' service	2,400
Past Assistant Surgeon (Captain)	
Over 17 years' service	$3,000
Over 7 and less than 17 years' service	2,400
First appointment above second lieutenant	2,400
Present rank July 1, 1920, or earlier	2,400
Less than 7 years' service	2,000
Assistant Surgeon (First Lieutenant)	
Over 10 years' service	$2,400
Over 3 and less than 10 years' service	2,000
First appointment above second lieutenant	2,000
Less than 3 years' service	1,500

In addition to this base pay, there are also rental and ration allowances. These vary according to whether the officer has dependents. The sum for rental ranges from $480 a year for an assistant surgeon to $1440 a year for an assistant surgeon-general. The sum allowed for rations is $237.25 a year for one ration. The allowance for subsistence is based upon the cost of food in the calendar year 1922. If the cost of food in subsequent years should be reduced, the allowance for subsistence will be accordingly reduced. There is also an increase for length of service in the grade. Thus, a surgeon in the Public Health Service who has completed fourteen years of service and has dependents, would receive a base pay of $3000, rental allowance of $1200, and ration allowance of $711.75 (three rations allowed), a total of $4,911.75 a year. If he had twenty years of service he would get a base pay of $3,900, with the same allowances.

The annual base pay of the Surgeon-General of the Public Health Service is expressly set at $6,500.

Nurse Corps. The bill allows the following amounts for pay of nurses: During the first three years of service, $840; from the beginning of the fourth year of service until the completion of the sixth year of service, $1,080; from the beginning of the seventh year of service until the completion of the ninth year of service, $1,380; from the beginning of the tenth year of service, $1,560. Superintendents of the Nurse Corps shall receive a money allowance at the rate of $2,500 a year; assistant superintendents, directors, and assistant directors, at the rate of $1,500 a year, and chief nurses at the rate of $600 a year, in addition to their pay as nurses. Nurses are also given an allowance for subsistence, the same as officers.

This proposed legislation is reported to have met with approval by medical officers in the Army, Navy, and Public Service.

THE TUBERCULOSIS FIGHT.

THE fighting equipment against tuberculosis consists of more than 700 well-equipped sanatoria, thousands of enthusiastic workers, 1,200 tuberculosis associations, over 600 tuberculosis clinics and a large number of traveling dispensaries and clinics, thousands of tuberculosis nurses, several thousand open-air schools and fresh-air classes, and a thoroughly aroused public opinion on the need for the control of the disease.

The time will come when it will be a disgrace for an American community to have a death rate from tuberculosis of over 10 per 100,000 population.

A HIGH DEATH RATE IN SCOTLAND.

DURING the week ended January 21, 1922, 379 deaths from influenza were registered in sixteen principal towns of Scotland. The population of these towns collectively is given as 2,370,600. This gives an influenza death rate for the week, on an annual basis, of 8.33 per thousand population.

DR. WILLOUGHBY, Medical Officer of Health for the Port of London, reports that the number of rats destroyed in warehouses from October 23 to November 26, was 2,522. On vessels during voyages to the port, 1,055 rats were killed, and on vessels in dock, 2,024. Since February, 1921, the total number of rats destroyed amounted to 1,209,169.—*Medical Press and Circular*.

Obituary.

JOSEPH WARD BATTERSHALL, M.D.

Dr. Joseph Ward Battershall, a retired fellow of the Massachusetts Medical Society, and formerly in the British Emigration Service, died at his home in Attleboro, February 23, 1922, at the age of 78.

Dr. Battershall was born in Troy, N. Y., May 1, 1842, his father, Ludlow A. Battershall, a wholesale grocer, and his mother, Eustasia Ward, daughter of Joseph Ward. The son had his education in the schools of Troy, and his medical training at the College of Physicians and Surgeons, Columbia University, New York City, where he took his M.D. in 1874. He entered the Pacific Mail Service, spent the year 1876 as sanitary inspector in New York, and in 1877 was appointed surgeon to the Schoolship *St. Mary*. From 1878 to 1879, he was in the British Emigration Service, on vessels plying between London and Sydney, Australia; he also made a voyage to China as ship's surgeon. Dr. Battershall settled in Yarmouthport, Mass., in 1879, joining the State Medical Society in that year and moving to Attleboro in 1881 to spend the rest of his life. He had served Attleboro as a member of the board of health, and had been in active practice up to a year and a half ago.

In 1887, he married Mary H., daughter of Robert Wolfenden, a graduate of the Woman's Medical College of Pennsylvania, in 1879. She survives him, as does a son, Dr. Jesse Wolfenden Battershall, now school physician of Attleboro. Both mother and son are members of the Massachusetts Medical Society.

◆

Correspondence.

AN OSTEOPATHIC DIAGNOSIS.

The following letter has been received by a person who consulted an osteopathic physician who lives in this vicinity:

Dear Mrs. Blank:

The examination of your blood by the Abrams test shows the following:

Congenital syphilis—30 ohms.

Streptococcus infection of the left antrum, left ethmoid, right antrum, right ethmoid, and slight involvement of the right sinus.

Very slight gonorrheal reaction.

Colisepsis.

The osteopathic examination shows lesions (spinal vertebrae slightly deviated from their normal position) of the first cervical vertebra, a first, second and third dorsal vertebrae, third lumbar, fifth lumbar, a slight convex curve on the left from the seventh to the twelfth dorsal, a lesion of the left innominate bone.

These conditions are amenable to treatment by the Oscilloclast and osteopathic adjustment.

The fee for this work is three hundred dollars ($300.00), the minimum two hundred dollars ($200.00) payable in advance.

Yours truly,

◆

BOSTON LYING-IN HOSPITAL STAFF.

The staff of the hospital as appointed for the year 1922 is as follows:

Visiting Obstetrician, Franklin S. Newell; Assistant Visiting Obstetricians, James R. Torbert, R. L. DeNormandie, F. C. Irving, Foster S. Kellogg, J. B. Swift, Jr.; Out-Patient Obstetricians, D. L. Jackson, D. J. Bristol, Jr., T. R. Goethals; Assistant Obstetricians to Out-Patients, William B. Young, W. T. S. Thorndike, John Rock; Visiting Pediatrist, Oscar M. Schloss; Assistant Visiting Pediatrists, Richard S. Eustis, Warren R. Sisson; Pathologist, S. Burt Wolbach.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

Reported Week Ending March 4, 1922.

Disease	*Cases*	*Disease*	*Cases*
Chicken-pox	114	Pneumonia, lobar	310
Diphtheria	192	Scarlet fever	215
Dog-bite	4	Septic sore throat	4
Encephalitis lethargica	6	Syphilis	42
Epidemic cerebrospinal meningitis	1	Suppurative conjunctivitis	6
German measles	5	Trachoma	5
Gonorrhea	82	Tuberculosis, pulmonary	169
Influenza	904	Tuberculosis, other forms	22
Measles	537	Typhoid fever	8
Mumps	96	Whooping Cough	90
Ophthalmia neonatorum	15		

THE COMPOSITION OF THE NATIONAL HEALTH COUNCIL.

Members, Officers and Staff.

Direct Members. American Public Health Association, American Red Cross, American Social Hygiene Association, American Society for the Control of Cancer, Conference of State and Provincial Health Authorities of North America, Council on Health and Public Instruction of the American Medical Association, National Child Health Council, National Committee for Mental Hygiene, National Organization for Public Health Nursing, National Tuberculosis Association.

Conference Member. United States Public Health Service.

Coöperating Through the National Child Health Council. American Child Hygiene Association, Child Health Organization of America, National Child Labor Committee.

Officers. Livingston Farrand, M.D., Chairman; Lee K. Frankel, Vice-Chairman; S. J. Crumbine, M.D., Recording Secretary; Wm. F. Snow, M.D., Treasurer.

Staff. D. B. Armstrong, M.D., Executive Officer: James A. Tobey, Washington Representative; Lawrence Marcus, Executive Assistant; Helen B. Eveline, Secretary and Assistant.

National headquarters, 411 Eighteenth street, N.W., Washington, D. C.

New York office, with the Common Service Committee, 370 Seventh avenue.

INSTITUTE FOR NURSES.

In making the final arrangements for the Institute for Nurses of the Boston Tuberculosis Association, the date has been set for Tuesday, March 28, with morning and afternoon sessions at the Boston Medical Library. Dr. Hermann Biggs, State Health Commissioner of New York, will speak, the list presenting papers including Miss Mary E. Marshall of New York City, Secretary of the Nursing Division, National Tuberculosis Association; Miss Mary E. Edgecomb of Providence, Supervisor of Tuberculosis Work, Providence District Nursing Association; Miss Margaret G. Weir of Beverly, Executive Secretary of the Public Health Dispensary Commission; Dr. Sumner Remick, Director of the Division of Tuberculosis, Massachusetts Department of Public Health, and Dr. John B. Hawes, 2d, President of the Boston Tuberculosis Association. All nurses are invited.

BOOKS FOR REVIEW.

The Journal acknowledges the receipt of the following books for review:

What Is Social Case Work?—By Mary E. Richmond. Published by Russell Sage Foundation. 268 Pages. Price $1.

Therapeutics—Preventive Medicine—Practical Medical Series, 1921.—By Bernard Fantus and William A. Evans. Published by The Year Book Publishers, Chicago. 386 Pages. Price $1.75.

Abdominal Pain.—By Norbert Ortner. Published by Rebman Company, New York. 362 Pages.

Protein Therapy and Non-Specific Resistance.—By William F. Petersen. Published by The Macmillan Company, New York. 314 Pages. Price $4 50.

A Laboratory Manual for Comparative Vertebrate Anatomy.—By L. H. Hyman. Published by The University of Chicago Press. 380 Pages. Price $2.50.

The Mechanics of the Digestive Tract.—By Walter C. Alverez. Published by Paul B. Hoeber, New York, N. Y. 192 Pages. Price $3.50.

The Spleen and Some of Its Diseases.—By Sir Berkeley Moynihan. Published by W. B. Saunders Company, Philadelphia, Pa. 129 Pages.

Infant Feeding (4th Edition).—By Clifford G. Grulee. Published by W. B. Saunders Company. 397 Pages. Price $4.50.

Psychoanalysis: Its Theories and Practical Application.—By A. A. Brill. Published by W. B. Saunders Company, Philadelphia. 468 Pages. Price $5.00.

Clinical Electrocardiography. — By Frederick A. Willius. Published by W. B. Saunders Company, Philadelphia. 188 Pages. Price $5.00.

Diseases of the Skin (9th Edition).—By Henry W. Stelwagon and Henry K. Gaskill. Published by W. B. Saunders Company, Philadelphia. 1313 Pages. Price $10.00.

Lehrbuch der Grenzgebiete der Medizin und Zahnheilkunde.—By. Dr. Julius Misch. Volumes I and II. Published by F. C. W. Vogel, Leipzig. 672 Pages.

Foods of the Foreign-Born in Relation to Health. —By Bertha M. Wood. Published by Whitcomb & Barrows. 98 Pages. Price $1.25.

An Essay on the Physiology of the Mind.—By Francis X. Dercum. Published by W. B. Saunders Company. 150 Pages. Price $1.75.

The Habit of Health—By Oliver Huckel. Published by Thomas Y. Crowell Company, New York, N. Y. 128 Pages. Price $1.00.

The Intestinal Protozoa of Man.—By Dobell and O'Connor. Published by William Wood & Co., New York, N. Y. 211 Pages. Price $4.50.

NOTICES.

A meeting has been arranged by the Boston Association of Cardiac Clinics to be held at the Boston City Hospital, Thursday, March 16, 1922, at 8:15 p. m. Dr. W. Irving Clark, of Worcester, will speak on Heart Disease in Industry. Dr. Harold H. Brittingham, of Boston, will speak on Exercise Tests and Vital Capacity. The meeting is open to all interested in the problems of heart disease.

The Boston Orthopedic Club.—There will be a meeting of the Boston Orthopedic Club, on Monday, March 20th, at 8.15 o'clock, at the Harvard Club. Program: "An Outline of the Metabolism and Treatment of Arthritis from the Practical Standpoint," Dr. Ralph Pemberton, Philadelphia. The discussion will be opened by Dr. Roger I. Lee, Dr. George R. Minot, Dr. Robert W. Lovett, and Dr. Joel E. Goldthwait. A large attendance is requested. Frank R. Ober, Secretary.

Boston Medical History Club.—There will be a meeting at the Boston Medical Library, Friday, March 17, at 8.15 p. m. Papers: Mr. Alfred Ela, "Protracted Pregnancies"; Dr. Frederic T. Lewis, Anatomical Notes, (1) Some Pregnancies not Protracted (Pinæus), (2) The Galenic Description of the Structure of Blood-vessels, (3) The Significance of the term Hippocampus (Arantius). At the annual meeting the following officers were elected: Dr. John W. Farlow, President; Dr. Malcolm Storer, Vice-President; Dr. John W. Cummin, Secretary-Treasurer; Mr. James F. Ballard, Curator.

The Boston Tuberculosis Association is planning an Institute for Nurses on the same plan as the recent one by this Association, for Physicians. It is intended to give to the nurses the latest facts with reference to the care of patients with tuberculosis, and speakers from outside the State will supplement home talent. The Institute will be in the hands of a committee including Miss Zepha M. Gardner, Superintendent of Nurses, Boston Consumptives' Hospital, Out-Patient Department; Professor Anne Strong of Simmons College, and Miss Bernice W. Billings, Executive Secretary of the Association. A location central in Boston will be selected for the Institute, the date will be Tuesday, March 21, subject to possible change, and the sessions will be in order both morning and afternoon. 7-3t

Children's Hospital.—Clinical Meetings of the Staff of the Boston Children's Hospital will be held in the amphitheatre once a month from November to May inclusive. The meetings will be held on Friday afternoons at 4.30 P.M. All members of the profession are cordially invited to be present. The dates of the meetings are November 4th, December 9th, January 13th, February 10th, March 10th, April 14th, and May 12th.

IMPORTANT NOTICE.

Announcement of meetings to be held on and after next Thursday should reach the desk of the Editor of the Journal not later than next Saturday before noon. The printers do not work Saturday afternoon and the material is locked up in the forms on Monday, and goes to press Tuesday morning The wrapping and mailing begins Wednesday. Please forward copy early.